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OTOGENIC COMPLICATIONS. A RESUME AND DISCUSSION OF THE LITERATURE FOR 1938.

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By the term otogenic complications are commonly understood those forms of infection which, originating in the middle ear, or its adjacent mastoid structures, by extension come to reach either: *1.* the meninges and subarachnoid spaces; *2.* the labyrinth; *3.* the brain; *4.* the lateral sinus and its various subdivisions; and *5.* the petrosal pyramid. In summarizing the rather voluminous literature on the subject of such complications it is convenient to subdivide them according to the particular region thus affected.

MENINGITIS.

The numerous articles available on this particular subject bear vigorous testimony to the remarkable progress made in the treatment of this heretofore almost hopeless condition, primarily through the utilization of the new drug, sulfanilamide. Case after case appears with reported recoveries in which the vital factor utilized in conjunction with appropriate surgery appears to have been this heaven-sent drug. As a result of this, there has been a renewed interest in the study of otitic meningitis as a whole, and a greater effort has been made to solve the problem of early diagnosis and ideal surgical treatment. Dwyer[1] considers as a primary object of surgery the removal of the focus of infection. Diagnosis is all-important in the decision as to whether meningitis is present and, if so, of what nature. The latter question is best answered by an examination of the spinal fluid, which should be physical, chemical, cytological, sero-

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logical and bacteriological. The fluid must be examined as soon as possible and placed in a tube sealed with melted paraffin after first dividing it into two parts, one for bacteriological and the other for a clinical examination.

1. The physical examination includes estimate of color and turbidity. Cloudiness is due to tissue reaction and the exudation of cells. If the organism simply irritates the subarachnoid space, there appears an exudation of leukocytes and a cloudy fluid, but no bacteria until the latter break through into the subarachnoid space.

2. The chemical examination includes an estimation of the amount of carbohydrates, the amount of globulin, the amount of lactic acid.

3. Cytological examination estimates the number of cells and their characteristics, whether lymphocytes or polymorphonuclear leukocytes. The cellular examination is of particular help in estimating the presence of tuberculous or syphilitic meningitis. Tubercle bacilli may be detected by a special technique, and serological tests are appropriate in detecting syphilis.

4. The bacteriological examination is important to determine the exact nature of the organism if present. It is essential to include meningococcic infection in consideration of otitic cases since it is, of course, nonoperative. To avoid failure in cultivation of the organisms, 1-2 cc. of a centrifuged specimen should be planted on appropriate media. Examination of the blood shows a high leukocytosis in meningitis together with a high polymorphonuclear percentage. Meningitis may be of a fulminating, acute, subacute or chronic type and may be the resultant of infection in such places as the petrous pyramid, which has been reached by blood stream infection rather than by direct continuity.

Friesner and Rosenwasser[2] feel that any operation designed for the sole purpose of drainage of the cerebrospinal fluid system must necessarily fail. Streptococcal exudate may appear in the walls of the basal cistern even when this is effectively drained, and it must be always remembered that meningitis may be an incidental and not an etiological factor. Operation should not be delayed if otherwise indicated simply because of the lack of definite findings in the spinal fluid. The authors have found petrous pyramid suppuration to be

the most common pathway of extension to the meninges. They prefer to divide the cases into meningitis which is secondary *a.* to infection in the labyrinth; *b.* to sinus thrombosis and phlebitis; and *c.* to infection of the middle ear, mastoid and petrous pyramid. In infection of the labyrinth, prompt labyrinthectomy is indicated with inspection of the dura of the middle and posterior fossae. The mortality is higher in acute than in chronic cases and repeated examinations of the cerebrospinal fluid are indicated. Where meningitis originates from a sinus thrombosis or phlebitis, it is important to get widely beyond the limits of the disease, both proximally and distally, and to lay the sinus widely opened. If the bulb is thrombosed, it should be irrigated with Dakin's solution, a procedure possible by the introduction of a soft catheter through the horizontal portion of the sigmoid into the bulb. In meningitis originating from infection of the middle ear, mastoid or petrous pyramid, a complete mastoidectomy is first indicated. Great care should be taken in searching for leads into the petrous pyramid, peribulbar cells and zygoma, and in the region of both the superior and posterior semicircular canals. The authors see no object in opening the dura.

Kopetzky[3, 54] has long been known as a tireless and productive worker in the field of otitic meningitis. In a general consideration of this subject, he comes to the following conclusions: Surgical drainage of the meninges is not the answer to the problem because of the multiplicity of foci spreading along the route of many pial vessels. The cerebrospinal fluid must be kept circulating and anything preventing this defeats the objective. Repeated small blood transfusions are the best means of maintaining a normal chemistry of the spinal fluid and promoting its active circulation. Best results occur in those cases in which the clinical picture is not too stormy and where pressure signs predominate over those of infection. Kopetzky attaches great importance to the removal of any bony focus surgically by breaking up the continuity of the skeletal dural vessels and thus interrupting infection which may pass along them. Like many other authors, he attaches great importance to a complete study of the spinal fluid. This includes not only an estimate of the amount of carbohydrate and sugar-reducing substances present and the amount of globulin and albumin in the fluid, but also an estimate of the lactic acid content as compared with that of the blood. Normally, there is a ratio between these of one to one, but in

meningitis the lactic acid ratio in the spinal fluid and in the blood will frequently be three to one. Whereas there are normally more chlorides in the spinal fluid than in the blood, in meningitis this relationship is more nearly equal. There is also a decrease of meningitis in the bicarbonate content of the spinal fluid. The Ph of meningitic spinal fluid is lower than normal, and is lower in the spinal fluid than in the blood. Kopetzky feels that it is important to realize that not all spinal fluid changes indicate meningitis. Often in uncomplicated otitis and acute mastoiditis there will be some rise in the cell content of the spinal fluid, but the chemistry is normal, there are no bacteria present, and the pressure is either normal or only slightly elevated. A weak protein reaction may possibly be found. With epidural suppuration there may be an increased number of cells but the chemistry is normal and the fluid again abacterial. In uncomplicated petrosal apicitis, there may be an increase in the cell count, and in sinus thrombosis, 50 per cent of the cases may show an increase in spinal fluid leukocytes, with polymorphonuclears often predominating and with some chemical abnormality. In the presence of brain abscess there may be a high spinal fluid cell count, abnormal chemistry and many bacteria unless the abscess is well walled off, in which case the fluid may be clear, with normal chemistry and an absence of bacteria, but with frequently an increase in pressure. The appearance of turbidity means that the abscess has ruptured or is leaking into the subarachnoid space. All these findings are evidence of meningeal invasion. Globulin and albumin will be found in traces; there will be a slight decrease in the amount of carbohydrate present; chlorides, carbonates and Ph are usually normal. The lactic acid ratio may be slightly increased and the clinical picture may be stormy, but there is as yet no evidence of true meningeal tissue reaction. The case is still one of meningeal invasion.

A true meningeal infection is indicated by gradual diminution of the carbohydrate contents, the positive presence of pathogenic bacteria, the gradual drop of the Ph of the spinal fluid, of the chlorides and the carbonates. The lactic acid ratio as compared with the blood gradually approaches three to one. More and more albumin and globulin are present. Spinal fluid becomes turbid or slightly purulent and the pressure rises. The clinical signs of headache, photophobia, etc., begin to appear.

The principal indication is a careful search of every portion of the temporal bone to locate the focal lesion which Kopetzky feels may often lie in the petrosal pyramid. Many cases may be seen too late to be amenable to surgery. He advocates the removal of all bone on the visceral side of the mastoid process, including the tegmen celluli, the tegmen tympani, and even the mesial wall of the petrosal pyramid. The author is a strong advocate of the use of sulfanilamide as better than any serum or vaccine. He warns against the formation of metahemoglobin, which he feels is best combated by the intravenous injection of 1 per cent solution of methylene blue in a dosage of 1 cc. per kilo of body weight. He has found this drug also effective in type III pneumococcal infection. He stresses the importance of frequent sulfanilamide estimates in the blood and describes a technique requiring only a few drops of blood for its execution.

Yaskin[4] encountered 123 cases of bacterial meningitis in 17 years, of which 37 were the result of infection in the temporal bone. One-half of these latter were due to the hemolytic streptococcus. The author stresses the importance of differentiating this type of meningitis, both from meningococcic meningitis and tuberculous meningitis. Moreover, he feels that bacterial meningitis may not infrequently be confused with such conditions as cervical adenitis, retropharyngeal abscess, meningismus, virus disease, hemorrhagic conditions, syphilis and aseptic meningitis, and that it may at times appear as a meningeal syndrome, etiologically unrelated to the ear and hence requiring quite different and appropriate clinical attention.

Meningitis of pneumococcic origin has always held for the clinician an outlook of the utmost gravity, particularly before the advent of sulfanilamide and its derivatives. Garfin[5] stresses the often mild initial symptoms of a pneumococcus otitis and the possibility of a prolonged period of remission before the advent of subsequent mastoid complication. He reports a case of a boy, age 10 years, who was the first on record at the Boston City Hospital to recover from an otitic meningitis due to the type III pneumococcus. He feels that recovery was greatly aided not only by sulfanilamide but by the use of an antibody in the spinal fluid prepared as follows:

1. Complete and frequent drainage of the spinal fluid.

2. Rapid identification of the pneumococcus and its type, and the intravenous administration to the patient of a sufficient amount of specific antipneumococcic serum to establish a balance of antibodies in the blood.

3. A fluid intake by whatever route is necessary to secure adequate amounts of spinal fluid for drainage.

4. Removal of from 5 to 10 cc. of the patient's blood, with rapid separation of the serum; and

5. Removal of spinal fluid and intraspinal injection of this immune serum. This procedure is repeated at subsequent lumbar punctures until the fluid returns to normal. Transfusions were given after the first week of sulfanilamide therapy to combat anemia, and were repeated until the drug was discontinued. This method was supplemented by a radical mastoidectomy and by the use of sulfanilamide, but it was noted that, in spite of clinical improvement, the spinal fluid did not become sterilized until after the intraspinal injection of the patient's own serum, which had developed antibodies for the type III pneumococcus.

The literature of the past year is replete with reported recoveries in authenticated cases of otitic meningitis after the administration of various forms of sulfanilamide. From these reports, in spite of the accessory therapeutic procedures, such as varying types of operation and other forms of medical treatment, it is manifest that this drug is fast becoming a boon to the otologist and permitting him to save the lives of patients in a manner formerly quite impossible. Granted that the drug has potential dangers and must be administered intelligently and cautiously, nevertheless, the following summaries indicate quite clearly the tremendous field of usefulness which it is fast coming to occupy in the treatment of this heretofore well-nigh hopeless condition.

Jackson[6] reports the case of a girl, age 6 years, who developed acute otitis media, which was followed by mastoiditis, for which she was operated upon. A week later there developed pain in the ear, vomiting, right external rectus weakness, but no elevation in temperature. Two days later there was right frontal headache and reflexes were increased. This was shortly followed by stiffness of the neck, positive Kernig sign, 6,000 cells in the spinal fluid, from which hemolytic

streptococci were later cultured. Patient was put on a Bradford frame, with continuous intravenous drip of glucose and spinal drainage every two hours. Intensive sulfanilamide therapy was started. X-ray showed a lesion of the petrous pyramid anterior to the superior semicircular canal. At subsequent operation an infected cavity was found just anterior to this point. Drainage of this was followed by complete recovery with normal hearing.

Cline[7] recounts the case of a patient, age 5 years, who, following an acute otitis media and simple mastoidectomy, developed signs of acute labyrinthitis in 48 hours. The patient was treated with sulfanilamide with temporary improvement, cessation of fever and disappearance of the nystagmus. Within the next few days there was an increase in headache, with vomiting and fever but no stiff neck or other positive signs of meningitis. Nevertheless, a lumbar puncture showed a cell count of 3,000, with hemolytic streptococci present both in smear and on culture. The author raises the interesting question as to whether the sulfanilamide given for the evident labyrinthitis masked the symptoms of meningitis and more particularly kept it localized. At no time was there stiff neck or positive Kernig sign. The subsequent treatment included a radical mastoidectomy with wide exposure of the dura and the administration of sulfanilamide intraspinally, orally and by clysis. This was followed by gradual improvement and a return to normal in the condition of the spinal fluid over a period of 10 days, with ultimate complete recovery.

Halphen[8] and his associates report the case of a boy, age 11 years, with otitic meningitis of a severe degree, with spinal fluid containing hemolytic streptococci, in which cure was obtained by massive doses of sulfanilamide, both by mouth and intraspinally. The authors stress the importance of surgical removal of a focus of infection in conjunction with the administration of sulfanilamide, the doses of which must be large and must be continued until all signs of cerebrospinal infection have passed. Otherwise, remissions are likely to occur. In spite of the large dosage, no untoward effects from the drug were noted. though the authors call attention to the customary precautions which must be taken in its administration. The fear of intolerance or complication or accident, however, must not deter one from giving the drug in adequate doses. While apparently not yet as popular on the Continent as in

this country, sulfanilamide is, nevertheless, being used more and more abroad.

Hubert[9] reports four cases of postotitic meningitis, three of streptococcic origin and one due to the pneumococcus, in which recovery followed the administration of sulfanilamide. In all these patients the organisms were identified in the spinal fluid. Previous to the use of this drug, in 100 cases the author had seen only one cure of similar infection. In one of the present cases the patient was almost moribund when treatment was begun, and in this instance intraspinal injections of sulfanilamide were added to the medications by mouth. In two other cases, frank meningitis was present on the second day, even before there was gross evidence of an otitis. The author suspects that the dosage was larger and more frequent than might have been necessary, but his elation in the complete recovery of these four patients is ample testimony to his belief that without the use of sulfanilamide not one of them would have recovered.

Recovery in what is supposed by Smith[10] to be the first case of meningitis due to anerobic beta hemolytic streptococcic infection is reported as due to the use of sulfanilamide. The patient was a girl, age 21 years, with an acute otitis media following a previous sore throat. Drum incision was followed by rapid improvement, with cessation of the discharge in 10 days and complete abatement of symptoms. Two weeks later there was an acute exacerbation with left aural discharge, supraorbital pain and left temporal headache. The next day there were signs of an acute meningitis, the patient was drowsy, toxic and had a temperature elevation up to 103°. A left simple mastoidectomy was carried out without exposure of the dura or sinus. Intramuscular prontosil was administered, with marked immediate improvement. Five days later, however, headache returned with a septic type of temperature, a stiff neck, diplopia and a positive Kernig sign. Thenceforth, 15 cc. of prontosil was given intramuscularly every eight hours. At this time the spinal fluid was cloudy, with 360 cells, an increased globulin and a positive culture of anerobic beta hemolytic streptococci. The prontosil was then increased to 30 cc. every eight hours, subsequently followed by the oral administration of 60 gr. a day. Following two transfusions, there was a rapid subsidence of all symptoms, with complete

recovery. The author stresses the importance of growing hemolytic streptococci under anerobic conditions.

Similar dramatic results from the treatment of otitic meningitis by the use of sulfanilamide are reported by Roberts,[11] Law[12] and by Martin[13] and Ellenberg.

A less common form of meningeal infection is that due to the influenzal bacillus, for which sulfanilamide has not heretofore been enthusiastically recommended. Gordon,[14] however, reports the case of a boy, age 7 years, who, following spontaneous rupture of the drum, developed in a week's time headache, nausea, vomiting, delirium and a temperature of 103°. The patient was stuporous, had a stiff neck, a positive Kernig reaction and a cloudy spinal fluid, with 390 cells, which on culture showed Gram negative bacilli. At operation, diploic cells were found to contain droplets of seropus in various areas. The lateral sinus was uncovered but found uninvolved, the dura was exposed and showed a suggestive inflammatory reaction. The patient recovered completely after lumbar punctures every eight hours and the administration of sulfanilamide, together with two transfusions.

In reporting the cure by use of sulfanilamide of a boy, age 6 years, with streptococcic meningitis, Sacks[15] stresses the importance of complete removal of the primary bony focus. He believes that all limiting bone plate should be removed from the zygoma anteriorly to and beyond the sinus posteriorly, and, likewise, that the nearest collection of cerebrospinal fluid should be evacuated by incision of the dura. This latter step in the operation has the support of many competent otologists, but, on the other hand, it is thought unnecessary by equally competent operators.

It is noticeable in the examination of many of these reports that sulfanilamide was administered intraspinally as well as by mouth. In a general discussion of the present status of sulfanilamide in the treatment of meningitis, Applebaum[16] expresses the opinion that there is no special advantage in the intraspinous administration of the drug. He feels that frequent small doses are equally as effective as less frequent large doses. Staphylococcic, influenzal and nonhemolytic streptococcic infections are far less amenable to sulfanilamide therapy, and in 30 cases of pneumococcic meningitis he had only four recoveries. None of these patients, however, was

treated with pneumococcic serum as advocated by Garfin.[5] In 26 cases of meningitis due to the hemolytic streptococci, there were 21 recoveries, the affection being due, in all but two, to an aural lesion. The amazing progress that has been made along these lines is clearly evident in the figures submitted, which show that prior to 1936 there had been only 15 reported recoveries in 274 cases of streptococcic meningitis. The author feels that subarachnoid drainage, repeated lumbar punctures and cisternal or ventricular punctures are fully as effective as laminectomy or trephining of the cisterna magna.

That even the most desperate cases of otitic meningitis will often yield to prolonged and intensive sulfanilamide therapy is evident by Diehl's[17] report of the case of a boy, age 15 years, with an acute otitis media, with spontaneous drainage continuing for three weeks. The patient gradually became stupid and irritable, complaining of severe headaches and exhibiting an elevated temperature, vomiting, and a left internal rectus paralysis and stiff neck. Spinal fluid was under increased pressure, was cloudy, contained 6,000 and later 9,500 cells with a positive smear for the hemolytic streptococcus. During the next month, patient was critically ill, underwent 19 spinal punctures, with a total removal of between 500 and 600 cc. of fluid, and received between 1,000 and 1,100 gr. of sulfanilamide, in addition to 800 cc. of transfused blood. His ultimate recovery was an illustration of what may be accomplished with this medication even in the most severe cases.

It has long been the observation of many otologists that middle ear infection following the operation for tonsillectomy tends frequently to progress to mastoiditis, sinus thrombosis or even more dangerous complications. Exemplifying this situation is the case reported by Smith and Scott[18] of a boy, age 6 years, who, following tonsillectomy, developed an acute otitis media. The next day he suffered a severe nasopharyngeal hemorrhage, requiring transfusion. Next in order followed fever up to 103° and the necessity for a right mastoidectomy. Following an improvement for six days with apparent recovery, fever recurred, with ultimate necessity of a left mastoidectomy. Following two weeks of improvement, fever recurred up to 103°, there was a stiff neck, positive Kernig sign and a turbid spinal fluid containing 2,300 cells with a positive culture for the hemolytic streptococcus. With surprising and yet

possibly commendable boldness, the authors carried out a double modified Neumann operation. The patient was supported with transfusions and intramuscular administration of prontosil. The drug was administered every day and lumbar punctures frequently repeated. Ultimate recovery followed and the patient now hears a whisper at 18 feet.

It has been manifestly impossible to study the effect of sulfanilamide experimentally on human beings with otitic infection. There has remained only the laboratory investigation with such animals as rats. As a result of their work with these animals, Adolph and Lockwood[19] feel that since streptococcic meningitis is a cellulitis of the meninges, the chemotherapeutic approach must logically be directed towards providing the meninges and underlying tissue with an environment unfavorable for their invasion by streptococci. They feel that this end can be achieved as well by giving sulfanilamide by mouth as by giving it intraspinally. The important factor is to obtain an adequate blood level. If, experimentally, the drug can be given coincident with the introduction of virulent organisms into the meninges and produce a typical alteration in the course of the disease, it might be expected to be even more effective in preventing infections spreading to this region. Hence, these authors feel that the practice of giving sulfanilamide to the patient with streptococcic otitis media and mastoiditis is to be strongly recommended, particularly when extensive surgical intervention may be required. This, of course, is at variance with numerous clinical opinions which express the feeling that under these circumstances the drug tends to introduce an element of confusion and to mask certain clinical symptoms which would otherwise be of great value in determining the course of the disease. These authors feel that whether the initial lesion is cured by the sulfanilamide is of secondary importance if further invasion is prevented. The drug does not act by promoting phagocytosis but by restricting multiplication of the organism and thus preventing invasion of the tissues and the blood stream.

These numerous reports of the great success of sulfanilamide do not, of course, shed any light on the possibility of cases equally well handled from a surgical and therapeutic standpoint in which, in spite of the adequate administration of sulfanilamide, a fatality resulted. Whether such cases exist in any considerable number and have merely not found their

way into the literature cannot for the moment be determined. Doubtless they have occurred, but, on the other hand, it is quite manifest that with the use of sulfanilamide a success in the treatment of otitic meningitis with appropriate surgery is being achieved in a degree which up until the time of the introduction of this drug was absolutely unknown.

LABYRINTHITIS.

In marked contradistinction to otitic meningitis, the complication of labyrinthitis, if the paucity of its literature is any criterion, seems rather to be on the wane. This is due, no doubt, to the more effective treatment of acute middle ear and mastoid infections at a time when they are amenable to conservative surgery and before any extension to the labyrinth has taken place.

A most informative and lucid discussion of labyrinthine infection is that of Cinelli.[20] He considers the four prominent routes of labyrinthine infection to be: *1.* A fistula through the round window; *2.* a fistula through the oval window; *3.* a fistula through the bony capsule of the labyrinth; and *4.* extension through vascular channels. The common site for a fistula through the bony capsule is at the prominence of the horizontal canal, especially anteriorly. He believes that labyrinthitis occurs in only about 1 per cent of all cases of middle ear infection and that it is not uncommonly associated with aural polypi. He divides the various forms of labyrinthitis as follows:

1. Chronic Circumscribed Labyrinthitis: This condition may be caused by: *a.* Pressure atrophy or ulceration of the bone due to an accumulation of cholesteatoma. *b.* Purulent destruction or necrosis of the mucous membrane overlying the prominence of the canal with rarefying osteitis such as is seen in acute otitis or in an acute exacerbation of a chronic otitis. Erosion leads to a fistula and then to acute serous and purulent labyrinthitis, with its various acute exacerbations.

In this type, healing is the rule before the development of the purulent stage with usual involvement of the horizontal canal. Circumscribed labyrinthitis is more common in the canal than in the cochlea. A feature of circumscribed labyrinthitis is the retention of hearing, the attack lasting about a week and then passing into a chronic stage, at which time

the patient is usually seen. There may be attacks of intermittent vertigo. Functional examination of the cochlea reveals a conduction deafness. Rotation tests on the uninvolved ear are normal; on the affected ear show only a diminished nystagmus, with caloric tests practically always negative. The fistula test, if positive, is important. With repeated attacks, this condition may go on for years to eventuate in: *a.* Complete healing with connective tissue and bone formation, with normal cochlear and canal function intact; *b.* the development of a circumscribed lesion of the membranous labyrinth, with repeated attacks of vertigo; *c.* the development of a diffuse, manifest purulent labyrinthitis.

2. Diffuse Manifest Labyrinthitis; a. Serous Type; b. Purulent:

a. The serous type is an acute, nonpurulent inflammation of the labyrinth with hyperemia and diffuse exudation involving the membranous labyrinth. It is at first difficult to distinguish from the purulent form, but begins to subside within a few days, whereas in the purulent form the cochlear and vestibular reaction disappear completely. The average suppurative form is usually preceded by a serofibrinous form due to the irritative action of bacterial toxins from the middle ear.

b. Suppurative labyrinthitis involves the destruction of the nervous elements, the nerve fibres and the more resistant parts of the membranous labyrinth. Granulation tissue will be found invading the labyrinthine structures and attempting to raise a barrier against the spread of infection. In the rapid and more virulent form, this barrier fails, with resultant meningitis. The clinical picture is that of a horizontal and rotary nystagmus from all three canals, beginning on the affected side, later shifting to the opposite side, showing the death of the labyrinth. The patient is acutely ill, lying in bed with his eyes closed. He suffers a rotary vertigo which corresponds in type to the same plane as the nystagmus, and he feels as though objects are rotating about him. He tends to lie on the sound ear, usually suffers from nausea and vomiting. The temperature is not significant, usually not over 101°, and headache is not a prominent symptom. Patient should be left alone and no test tending to spread the infection should be carried out, at least until the patient's condition improves. In the latter event, one may do caloric and cochlear

tests, a negative response to which should awaken suspicion of a dead labyrinth. If the lesion is serous, hearing and vestibular reaction will reappear in three to four days. Barany is accustomed to wait eight days before calling the lesion purulent, in which case hearing and vestibular reaction will be completely absent. In six weeks the patient may feel quite comfortable and desire to get up, but he should be kept in bed three months. During this period the internal ear begins to fill with granulation tissue, which gradually changes to scar tissue and final calcification. Final healing occurs in about 50 per cent of all cases of purulent labyrinthitis. Labyrinthitis occurring during the first 12 days in an acute otitis media is usually serous; if coming on in the third week or later, usually purulent.

Serous, diffuse manifest labyrinthis usually eventuates in a normally functioning labyrinth, whereas the purulent type may end with a completely ossified and functionless labyrinth, if not in intracranial complications.

3. Diffuse, Serous Induced Labyrinthitis: This type differentiates itself from diffuse manifest serous labyrinthitis; first, because the latter usually complicates a chronic, circumscribed labyrinthis; second, because it has prodromal vestibular symptoms; and third, because the fistula test is always present. In the former, or induced type, there is no relation to a chronic, circumscribed labyrinthitis; there are no prodromal vestibular symptoms, the onset being sudden; and the fistula test is always negative. It usually occurs either following an acute purulent otitis media or in cases of an unhealed radical mastoid operation or a short time following the radical operation, usually one or two days later, quite suddenly, just as the patient begins to feel better.

4. Latent Purulent Labyrinthitis: Here there is usually a history of an attack of previous diffuse, manifest purulent labyrinthitis. The patient may at the moment feel fine, with no symptoms except a chronic running ear with deafness. Functional tests show this to be complete and indicate an absence of the caloric reaction without fistula symptoms. If there is still a reaction indicating vestibular activity on the diseased side, final ossification has not yet set in. Ultimately the good ear will assume all the function from the affected side and thus compensate for the unilateral lesion.

In an article discussing the clinical aspects of labyrinthitis, Wails[21] considers that retrograde labyrinthitis secondary to an already existing meningitis is a practically hopeless condition. If, however, labyrinthitis starts from a middle ear infection, it is of the greatest importance to check it before it creates a purulent meningitis. Spontaneous nystagmus, severely impaired hearing, marked tinnitus and vertigo would all be suspicious of a labyrinthine complication.

Circumscribed labyrinthitis may be traumatic, serous or purulent. It may affect only the bony capsule and irritate the endothelial membrane by continuity or the latter may actually be invaded. Treatment of a circumscribed labyrinthitis, if too vigorous, may easily convert it into an acute form as, for instance, in a radical mastoidectomy. Serous labyrinthitis may simulate Mèniére's syndrome and is often the result of a systemic toxicity or focal infection. Purulent labyrinthitis may be the result of trauma or an extension of a circumscribed labyrinthitis. Initial signs are those of marked labyrinthine irritability with nausea, vomiting, deafness, tinnitus and headache. With gradual death of the labyrinthine structures, the activity of the good ear predominates and the nystagmus will be to the side of the latter. Serous labyrinthitis secondary to acute otitis media may often be cured by a simple mastoidectomy, that secondary to chronic otitis media by radical mastoidectomy. If the latter fails, opening of the labyrinth is then indicated. Purulent acute labyrinthitis will always be manifested by some irritation of the meninges with a cloudy spinal fluid and increased cell count and a reduction in the carbohydrate contents. Organisms may or may not be present. Even then a radical operation is not necessarily indicated if a delay shows improvement in the condition of the spinal fluid. The picture is that of a circumscribed meningitis and simple drainage of the labyrinth may be all that is indicated.

Bradbeer[22] cites an interesting case of acute labyrinthitis in a woman, age 24 years, with an acute right otitis media complicating influenza. The patient complained of right aural discharge, vertigo and vomiting, but no fever. There was rotary nystagmus on looking to the left, profound deafness in the right ear and hemorrhagic blebs in the external meatus. Following an incision of the drum with escape of pus from the left ear under great pressure, no caloric reaction could be

obtained from either ear. In spite of subsidence of dizziness, nystagmus and discharge from each ear, the right still remained absolutely deaf and the caloric reaction was unobtainable from either side. In a few days, however, caloric stimulation of the left ear was effective and ultimate recovery took place. This patient undoubtedly suffered destruction of the right labyrinth but the author raises the interesting question as to why no caloric response was temporarily obtainable from the uninvolved labyrinth until symptoms of acute destruction of the other labyrinth had subsided. For this unusual phenomenon he offers no explanation.

BRAIN ABSCESS.

A most interesting statistical study of brain abscess is reported from Sercer's[23] Clinic in Zagred, in Jugoslavia. From 1922 to 1937, there were 32 patients with 34 brain abscesses; 18 cerebral and 16 cerebellar. In some years not a single case was seen; in others, three or four. The author is at a loss to explain the varied incidence of brain abscess in different places. In his series there was one brain abscess to 76 patients hospitalized for middle ear inflammation, whereas in Prague there was one in 506, in Munich one in 1,764, and in Budapest one in 1,000, a difference possibly due to the fact that the author included only middle ear inflammations that were hospitalized and not ambulatory patients. Also, it is probable that in many places where the public had access to clinics, the number of simple middle ear infections was proportionately greater than in a small clinic, where only the more complicated cases tend to be seen. At autopsy, he also found a similar ratio, there being in Zagred one abscess in 393 autopsies, in Vienna one in 435, and in Munich one in 784. In Sofia, in Bulgaria, however, one clinic saw 40 abscesses in 10 years. In the author's series of 34 abscesses, 23 occurred in men, 11 in women, a ratio of two to one, which is frequently found in other places. The commonest age group was that between the second and third decades of life. Whereas, in Budapest, cerebral abscesses were found quite as commonly as cerebellar abscesses, in the author's series they were divided about half and half. Of the cerebral abscesses, 12 were on the right, six on the left; of the cerebellar abscesses, seven on the left, nine on the right. There were two instances of bilateral abscess.

Etiologically, four cases followed acute, and 28 cases followed chronic middle ear infection, 16 of the latter showing cholesteatoma. In other clinics, cholesteatoma was still more common, amounting in Munich to 87 per cent of the total number. In 13 of the author's cases there was associated sinus thrombosis, and in nine a labyrinthitis. Only 22 per cent of the author's patients recovered, death being due in 19 to meningitis, in two to pneumonia, in two to lung abscess, in one to encephalitis and in one to cavernous sinus thrombosis. Four of the patients were moribund when brought into the hospital.

Attention is called to the fact that the cured patients, if watched long enough, may still show certain residual symptoms, such as blindness, paralyses, epileptiform attacks, psychic disturbances, poor memory, hypersensitiveness, etc. The prognosis appears to be better in otogenic than in traumatic brain abscess. Of those patients who recovered, it was found that symptomatically a slow pulse had been present in only a few instances, that all had had headache, and that vomiting was common in cerebellar but not in cerebral abscess. In only three of the seven recovered patients was there a definite purulent reaction in the spinal fluid, and although the abscess occurred in three instances in the left cerebral hemisphere, only one of these patients showed any aphasia.

Two patients with cerebellar abscess had nystagmus before operation and in one this symptom appeared after operation. In one instance an abscess herniated spontaneously as a subperiosteal abscess, in another fatal instance the abscess broke spontaneously into the middle ear.

Treatment in the seven successful cases consisted of wide trephining and opening of the abscess, together, in one instance, with a sinus operation, and in another of drainage of the lateral cistern.

In all but one of the 25 fatal cases, headache was a marked complaint. There was vomiting in only six of the 13 patients who died of cerebral abscess, and in nine of the 12 who died of cerebellar abscess. Vomiting was a prominent symptom in six of the 17 patients with cerebral abscess, and in 12 of the 15 patients with cerebellar abscess. Slow pulse was found in only five of the 13 cerebellar fatalities, and in two of the 12 cerebral deaths.

Psychic disturbances, present in 12 of the patients, were twice as common in cerebral as in cerebellar disease.

In 11 of the patients who died of cerebral abscess, the spinal fluid was purulent and in three instances showed definite bacteria. Of the 12 cerebellar fatalities, spinal fluid was purulent in 11 and in one instance showed bacteria. In a total of nine left-sided cerebral abscesses, aphasia was noted only twice. In the cerebellar cases, nystagmus was present in 14 out of 15 cases. Discussing the pathology of brain abscess, the author feels that it starts as a primary encephalitis and that it is the progression of this encephalitis, in spite of incision and drainage, which brings about fatality. In general, the best track to follow in surgical treatment is that taken by the original infection, from the middle ear to the dura and brain. One may, however, be deceived by the appearance of the dura and thus misled as to the location of the abscess. It may develop very rapidly in a few days, particularly following exploratory puncture. Mixed bacterial infections are the rule in abscesses associated with chronic middle ear infections, whereas in the acute cases a single organism is usually responsible. A communication of an abscess with the ventricle may be due to the rupture of the abscess into the latter or of an accumulation of infected ventricular fluid into the abscess. The author does not favor the use of lipiodol injections in abscess cavities since he feels that this procedure is not without danger and that a delay in elimination of the material from possible pocketing of the abscess is disadvantageous. A correct diagnosis depends on the stage in which the patient is seen and the quality, extent and localization of the abscess, due consideration being, of course, given to any other complication which may be present, such as labyrinthitis, petrositis, sinus thrombosis and meningitis. The history and general symptoms, such as the blood picture, temperature reaction, psychic disturbances and X-ray findings, will frequently prove unreliable. The same is true of the generalized symptoms of increased intracranial pressure, which must be estimated with the greatest care. Many processes compressing the brain exhibit no signs of increased pressure, and others which do not compress it give very marked symptoms. Local evidences, such as aphasia, ataxia, adiadokokinesia, hypotonia, etc., are valuable but seldom sufficiently outstanding to be trustworthy. Nystagmus is the

most constant symptom in cerebellar abscess, the diagnosis of which, however, is still extraordinarily difficult. Of greatest diagnostic import are headaches, vomiting and the state of the spinal fluid, on which the author lays great stress. He expresses regret that the chemical analysis of the spinal fluid cannot be extended and more refined, and feels that medical intuition is still a most important diagnostic factor. In the present series, treatment consisted in trephining the mastoid process with a wide dural exposure at the appropriate place. In certain instances, either at the time or later, labyrinth and sinus operations, together with drainage of the lateral cistern, were carried out. The abscess was located by needle puncture and when found the abscess cavity was widely opened, drainage being effected by a rubber drain, glass drain, silver wire or iodoform gauze. All abscesses were approached through the old wound, never through the temporal squama or occiput. The Killian speculum was found very useful for inspecting the interior of the abscess cavity, which was cleaned out with great care and dressed often twice a day. Repeated lumbar punctures aided greatly in reduction of intracranial pressure and facilitation of drainage. In spite of all efforts, a large number of patients died as a result of progressive encephalitis or meningitis.

The author states that he has never been able to follow the delayed type of treatment advocated by Dandy, or grossly to enucleate an abscess as one would a brain tumor.

He stresses the importance of general care of the patient, particularly of forcing fluids by all means. He prefers progression in stages, seeking first an extradural abscess or infection or uncovering the dura and waiting for further signs and symptoms if an abscess is present, and for adhesion of the dura at this point. He feels that his failures were primarily the result of inability to drain the abscess sufficiently to stop the progress of the surrounding encephalitis. Much depends upon the intensity of the biological defense reaction in the brain substance and the virulence of the infecting organism. Possibly some form of intracarotid injection of an appropriate medication might prove helpful and capable of stimulating a defense reaction in the brain tissue.

Meurman[24] summarizes his personal experience with brain abscess between 1901 and 1937. There were 77 infections, of

which 13 were acute and 64 chronic. Of the acute infections, three were in the cerebellum, 10 in the cerebrum. Of the chronic, 32 were in the cerebellum, and 30 in the cerebrum. Taking both groups together, they were thus about equally divided between the cerebrum and cerebellum. Of the cerebral abscesses, of which there were 40, 25 were single and seven multiple, though not clinically metastatic. The author feels that these may have developed from a single irregular cavity which later in some way became divided into multiple abscesses. Of the 35 cerebellar abscesses, 27 were single and six multiple. Of the 25 single cerebral abscesses, 16 were opened, nine were unopened, with cures obtained in only eight patients. Of the multiple cerebral abscesses, six were opened, one unopened, with four recoveries. Of the cerebellar abscesses, of which 27 were single, 18 were opened, nine unopened, with only nine recoveries. Of the multiple cerebellar abscesses, all six were opened, with two recoveries, indicating thus that the prognosis is much worse for multiple than for single abscesses. The author stresses the great use and importance of making use of encephalography and ventriculography and feels that a greater adaption of methods now commonly used by neurological surgeons should be in vogue with the otologists.

In this connection it is interesting to find that Laskiewicz,[25] in discussing the development of cerebral abscess, points out the fact that in many instances what appear to be multiple abscesses are in reality sacculations and lobulations of a single cavity which slowly advance into the white matter of the brain and, ultimately reaching the ventricle, provoke, by virtue of their highly virulent infective contents, a fatal meningitis. He pleads for a wider use of the instillation of opaque media into such abscess cavities in order better to delineate their subdivisions and loculations. Only a small quantity, from 2 to 3 cc., is necessary and he has never seen it exert an unfavorable influence on the evolution of the process. Rather, it seems sometimes to diminish the virulence of the organism in question. This method has many advantages. It shows the size and location of the abscess and, particularly where direct observation of the interior by means of a speculum does not permit determination with certainty of the presence of diverticula which may be progressing ever deeper, radiography with a contrast medium often visualizes these

and indicates their location. The presence of such digitations indicates that the purulent process is penetrating the cerebral tissue. Hence, the determination of their existence and the direction in which they are moving toward the ventricle is of prime importance in the diagnosis and treatment in the case under consideration.

Discussing the pathology of brain abscess, Brunner and Dinolt[26, 27] consider that the formation of a capsule of a brain abscess depends primarily on the cessation of contact with the middle ear infection, permitting the abscess to wall itself off by connective tissue reaction. During this latent stage of capsule formation, the abscess increases in size and new connective tissue is built around, which contains numerous blood vessels. This process may continue until surgical intervention or until the abscess ruptures into the ventricle or subarachnoid space or subcortically, or even into the middle ear, without necessarily producing any acute exacerbation of infection in the latter. The authors disagree with McEwen, who thought that certain abscesses may resolve and disappear. They believe that the chronic course in the brain abscess is manifested by an anatomic or clinical stage of latency, either complete or incomplete, which may last for several weeks or months. It is of secondary importance whether the abscess in this time has a capsule and whether it shows local symptoms or not. The significant thing is that during this period there is no anatomical or clinical sign of progress. Every encapsulated abscess must have had a latent period. but not every abscess which has passed through a period of latency will necessarily be encapsulated. Proper estimation of any psychic symptoms is most important in trying to establish the existence of an abscess in its resting stage. It is of less importance how such an abscess is opened as when it is opened. All varieties of operative technique have been followed by success but the prognosis is best when the abscess is opened in its latent stage.

The authors call particular attention to the necessity of differentiating between a true brain abscess and a phlegmonous encephalitis, with which such an abscess may readily be confused. A chronic latent brain abscess differs from a phlegmonous encephalitis in that the latter shows no resting stage and runs a very stormy course. Moreover, it shows a greater number of generalized and local symptoms, together

with a tendency to prolapse on incision of the dura. Needle puncture is frequently positive in brain abscess, but always negative in encephalitis. In brain abscess, the history will indicate long-standing general symptoms, such as headache and subnormal temperature, and meningeal symptoms are more prominent in many cases. The prognosis in both conditions is bad, but the usual treatment of an abscess in other parts of the body cannot be applied to the brain and half-way measures are useless. An acute abscess, in the true sense of the word, never goes into a long-standing latent stage nor has it a capsule, its margin being formed by a progressive purulent encephalitis. Acute abscesses, therefore, are the end-result of a diffuse purulent encephalitis, with single or multiple abscess formation. It is, however, true that a chronic abscess may be associated secondarily with a diffuse purulent encephalitis, particularly when emerging from its resting stage into a manifest stage. In this connection, as representative of a purulent, progressive or phlegmonous encephalitis, the authors cite the following two cases.

The first is that of a man, age 44 years, who was operated upon for simple mastoiditis. The sinus was uncovered but not opened, its walls being covered with granulations. There were subsequent signs of sepsis, necessitating a second operation, at which the sinus was opened and found to contain pus. Opening just posterior to the sinus knee and into its lumen was a mass of prolasped brain tissue consisting of hemorrhagic necrotic areas springing from the basal part of the lateral surface of the right occipital lobe. Subsequent needling of this prolapse revealed no abscess cavity. There followed later fixation of the pupil, a left-sided hemianopsia and death of the patient. This process, the authors believe, was due to a retrograde thrombosis of the pial and cerebral vessels. It was not a circumscribed encephalitis since it involved the entire occipital lobe and had a tendency to spread to the convolutions on the other side. Although there were small microscopic abscesses, the condition was essentially a diffuse phlegmonous encephalitis, rapidly progressive, without actual abscess formation or invasion of the ventricles. In this respect it was unlike either an acute or a chronic brain abscess.

In a second case, an acute exacerbation of chronic otitis was followed by intracranial complications. Radical mastoid operation showed a severe pachymeningitis. The dura was subse-

quently incised and showed a large, primary cerebral prolapse of the temporal lobe. Needling of this area revealed no pus. In this instance, too, in the absence of actual abscess or meningitis, the condition must be considered one of phlegmonous encephalitis. This resembles metastatic encephalitis, in that both forms can lead to abscess formation and break into the ventricle, and both show a rapid progressive course and a rich clinical symptomatology. Metastatic encephalitis, however, shows a greater degree of diffuse extension and no topographical relationship to the point of origin of the infection and is usually only one part of a generalized pyemia.

With regard to cerebral abscess and its pathogenesis, the authors believe that infection follows the veins into the gray matter, where a circumscribed purulent encephalitis is set up. Gradually, with extension and dissolution of the encephalitic focus, there develops a pus-filled cavity or abscess. There follows a latent period of varying duration, during which the abscess may or may not form a capsule. Unoperated upon, the pus-filled cavity is surrounded by a purulent progressive encephalitis until symptoms are manifest. It then either breaks into the ventricle or into the subarachnoid space with a fatal terminal meningitis. This is the typical chronic brain abscess. Chronicity presupposes a period of latency of varying duration and completeness. It is also possible for the initial circumscribed encephalitis to develop first rapidly and to proceed to the formation of one or more abscesses without a capsule which never undergo a state of latency. These are the typical brain abscesses.

Thus, initial circumscribed encephalitis may develop in three forms: either toward a chronic brain abscess, toward a pure phlegmonous encephalitis or toward an acute brain abscess. Variations of virulence of the organism and the patient's resistance and the type of infection are factors which determine in which direction the course will go. Of the three, phlegmonous encephalitis has the worst prognosis. The major therapeutic problem is the prevention of the spread of meningitis from the margin of the prolapse. The major indication is the adoption of the type of surgical attack best suited to the type of the abscess.

As far as the differential diagnosis goes, the presence or the absence of a capsule is of only secondary importance. The

fact that many abscesses are found with capsules at postmortem is no guarantee that they are chronic because operation on an acute abscess which later comes to autopsy may have been a potent factor in the formation of a reactionary capsule. Such abscesses would have to be classified actually as acute. Since most abscesses forming in connection with chronic suppurative otitis with cholesteatoma have no opportunity to wall themselves off, they are, properly speaking, acute abscesses. Many chronic abscesses are metastatic or traumatic in origin, quite unlike otitic abscesses. The authors do not agree with the American point of view of letting acute abscesses alone until capsule formation; first, because it is difficult to tell exactly in what stage the abscess is at any given time; second, because it is dangerous to await the appearance of a resting stage; and third, because it is possible to cure an acute abscess. That delay in waiting for capsule formation is dangerous is proved by the many autopsies on patients operated on for supposed sinus thrombosis or meningitis or apoplexy, the real cause of death being a brain abscess.

As to the ideal form of operation, many methods suit the chronic abscess but no specific method applies to the acute. A wide decompression of the brain is the principal thing. Theoretically, the brain itself should be disturbed as little as possible, although obviously many authors have succeeded with radical methods.

An interesting case is that reported by Tromeur,[28] of a patient, age 21 years, with an apparently simple furuncle of the canal complicating chronic otitis media following scarlet fever at the age of 15 years. Suddenly there developed headache, elevation of temperature and pain over the mastoid antrum, with profuse discharge. Simple mastoidectomy was undertaken, the mastoid containing hemolytic streptococci. Severe headache and fever followed, necessitating a complete radical mastoid operation, during which the patient temporarily collapsed on the table. After a general improvement of several days, sudden epileptiform attacks appeared, with marked neurological disturbances and with recurrences within a short space of time. The temporosphenoidal lobe was decompressed, with indications of intracranial increased pressure. The lateral sinus showing no evidence of pathology, the temporal lobe was punctured, with discovery of an abscess, into

which was placed a drain. The patient subsequently died, and autopsy showed a large abscess of the temporosphenoidal lobe. Moreover, the lateral sinus contained pus from the bulb to the torcula. The author calls attention to the marked latency of the abscess up to the time of the Jacksonian epileptic attack. In view of the history, this abscess was undoubtedly chronic and showed a thick capsule. There was nothing characteristic about the headache to point to an abscess and the patient himself made very little of it. He was, therefore, at the time of his admission in an asymptomatic or latent period, such as often antedates a severe complication. There was no reason to think that the mastoid operation would not be amply sufficient since there was marked temporary improvement with disappearance of the headache. Epileptiform attacks made the diagnosis of cerebral abscess a possibility, though these are not a common symptom. There was nothing else clinically to suggest a brain abscess beyond possibly striking and gradual emaciation of the patient, a symptom which has been thought by many to be quite characteristic of a brain abscess. In many ways it paralleled the headache but instead of being progressive or constant, it had appeared with alternations of aggravation, cessation and regression, and had previously brought up the question of a possible tubercular lesion. Although this patient showed clinically cholesteatoma, there was no macroscopic break in the tegmen or antrum of the middle ear. The final unusual circumstance in the case was the development of a sinus thrombophlebitis, which appeared to be the ultimate cause of death. This complication is rare in encapsulated brain abscess.

The essential features here are, first, the short duration of actual signs of an abscess; second, the onset with epileptic attacks; third, the lack of symptoms previously sufficient to make the diagnosis apparent; and fourth, the complication of sinus thrombosis. The latter illustrates the importance of exploration of the lateral sinus in any case of brain abscess in which, in the face of good drainage, temperature remains elevated, even if blood cultures are negative on several occasions.

In spite of the increasing frequency of its use in the treatment of meningitis, there has as yet been little written about the use of sulfanilamide in the treatment of brain abscess. Of

great interest, therefore, is the report of Bacy,[29] of the case of a girl, age 4 years, with right otitis media. Following a three weeks' improvement, there developed pain, vomiting, inability to walk, retraction of the head to the left, bilateral papilloedema and deviation of the eyes to the left. Voluntary movements were ataxic and the diagnosis was made of cerebellar abscess. There were obvious signs of increased intracranial pressure, somewhat relieved by ventricular tap. A suboccipital exploration was carried out, the dura was incised, the left hemisphere disclosed. On further exploration of the cerebellum, an abscess was encountered in the right hemisphere, and by suction a quantity of pus was removed which showed on culture hemolytic streptococci. During needling, the puncture wound was obviously contaminated and meningitis seemed unavoidable. Intensive sulphanilamide therapy was immediately instituted, the abscess itself was never drained, superficial drains being placed only near the region of the puncture wound. Recovery was complete and rapid. An unusual clinical feature was the conjugate deviation of the eyes away from the side of the abscess with inability to bring them back beyond the midline. The author feels definitely that the sulfanilamide prevented the development of meningitis. The abscess apparently was cured by a single aspiration, a unique experience so far as this author is concerned. Sulfanilamide undoubtedly had something to do with the prevention of refilling of the abscess. In this connection, the author outlined his customary treatment as follows: A 3-4 cm. defect is made in the skull overlying the abscess, the dura is incised and subdural space above the opening is packed with gauze soaked in a weak solution of iodine. If the intracranial tension is high, a needle is inserted and pus is allowed to escape. Six days after the first operation, the wound is reopened, the abscess widely exposed, evacuated and drained. The author feels that in the future, at the time of the first operation, if the organism proves to be a hemolytic streptococcus, he would administer sulfanilamide. If, under this treatment, the abscess does not refill and the patient continues to improve, he would undertake no further surgical treatment. If the abscess does refill, drainage can be carried out by the second operation as above. He stresses the importance of watching for toxic signs of the drug and of administering transfusion or discontinuing it if necessary.

Discussing the lack of unanimity as to the best treatment of brain abscess, Guns[30] quotes the gloomy feeling expressed in 1927 by Beck and Pollack, relative to the prognosis in brain abscess, and the fact that at Neumann's Clinic, since 1919, of 40 brain abscesses there has been recovery in only 12. A preoperative diagnosis, however, had been made in 37 of these 40 cases. From other reports, it would appear that the ideal method of treatment is the utilization of puncture in acute brain abscess while awaiting the formation of a capsule, following which enucleation of the abscess is undertaken in much the same manner as in the case of a cerebral tumor. Simple repeated puncture, in favor with other operators, has apparently only given temporary results. Guns cites two cases of brain abscess, one following chronic otitis media, the other acute. In the former, in a child, age 6 years, who had suffered from otitis media since the age of 6 months, a brain abscess was discovered at the time of a mastoid operation. Repeated evacuation of the abscess by needle puncture was combined with repeated lumbar puncture. The patient's condition becoming worse, a drain was inserted into the abscess cavity. Lumbar punctures were continued and the patient gradually made a complete recovery without any further surgical procedure. The author feels that an important prognostic sign lies in a study of progressive changes in the blood.

In the second case, with an extradural abscess and abscess of the temporosphenoidal lobe, the latter was treated by aspiration and insertion of a filiform drain, with subsequent incision of the meninges and placement in the cavity of the abscess of a drain of iodoform gauze. This treatment was continued for six weeks, with ultimate complete recovery.

As a result of these experiences, the author feels that enucleation of an encapsulated abscess is not always necessary and that puncture, reinforced with drainage, will often prove sufficiently effective if combined with a large decompression over the abscess. He believes, in fact, that enucleation should be reserved only for exceptional cases.

SINUS THROMBOSIS.

In an exhaustive study of sinus thrombosis based on 343 replies to a questionnaire sent all over the country, Evans[31] finds the general incidence of thrombosis to be 2.6 per cent,

with wide variations according to individual reports, some being as high as 7 per cent, others as low as 1 per cent. In almost 1,000 cases of sinus thrombosis, there were about 300 deaths, or a mortality of 33 per cent. Much depends on the question of inclusion of desperate or even hopeless cases in any given series. Most deaths were due to complications, such as meningitis or an intracranial spread of the infection, to pulmonary complication or distant metastatic lesion in vital organs. The usual bacterial organism was the streptococcus hemolyticus, Friesner obtaining positive blood cultures in 90 of 100 cases of lateral sinus thrombosis. The next commonest organism was the pneumococcus type III.

The symptomatology is varied and there is no single classical combination, particularly with regard to temperature curves, as has already been stressed by Tobey. A choked disc is of little definite diagnostic significance, but edema or infiltration with tenderness on deep pressure over the area drained by the emissary vein is an important objective symptom. Evans calls attention to the opinion, erroneously held, that a normal exposed lateral sinus pulsates. A thrombosed sinus may not pulsate but if pulsation is present, thrombosis is also probably present. A positive blood culture is a most important diagnostic sign but its absence does not exclude thrombosis. Cultures are more likely to be positive if blood is obtained at the beginning of a chill.

Reports indicate that the Tobey-Ayer test for unilateral thrombosis is of great value but not infallible, since anatomical abnormalities would obviously interfere with its accuracy.

In a series of 43 answers as to details of treatment, exposure of the sinus with ligation of the jugular vein was reported in 21. Many authors favor resection of the vein, while others express equal enthusiasm for simple ligation. Those opposed to ligation stressed a retrograde development of the thrombus and the great importance of supportive medical treatment, with transfusions, sera, forced feeding, tonic, ultraviolet irradiation, and such drugs as sulfanilamide. Ligation in their opinion does not prevent metastasis, embolism or septicemia.

A difference of opinion was evident with regard to the efficacy of transfusions, as to whether they definitely influence a

septic temperature and have any other supportive effect than the contribution of nourishment to the patient without in any way stimulating the formation of antibodies. The report showed sulfanilamide to be now riding a high wave of popularity in the treatment of sinus thrombosis with apparently excellent results. Due consideration must, however, in the opinion of the reviewer, be given to its disadvantages and undesirable reaction, not to mention its masking effect, particularly in the earlier stages of the infection. It is generally agreed that the drug must be continued for some time after apparent convalescence is begun and that surgical drainage of an infected thrombus as early as possible is still an important factor in the successful management of the disease.

Juers,[32] in a general consideration of otitic sepsis, believes that in any blood stream infection secondary to middle ear or mastoid disease, phlebitis at least is always present, and that thrombosis may or may not be. In a given series of patients he found chills more frequent in patients over 15 years of age, and none in children under 10 years. He feels that there is no relationship between a positive blood culture and chills, the latter being absent in four cases with positive culture. Severe sepsis is often present without an elevation of the white blood count. Surgically, a wide exposure of the sinus is a prime essential. If a thrombus is present, its complete removal is indicated to obtain free bleeding both above and below, in the attainment of which a suction cannula is very useful. Excision of a diseased sinus wall may be indicated. With regard to the much discussed ligation of the jugular vein, he noted that in 50 instances of negative blood culture before ligation, 15 cultures were found positive after ligation. The author classifies his own cases as follows:

a. Simple Phlebitis: Of these there were nine, of whom seven recovered with complete mastoidectomy and exposure of the sigmoid. In this group there was one death even after ligation, and another death from cerebellar abscess and meningitis.

b. Mural thrombosis, of which there were five instances. Four patients recovered completely after primary obliteration of the sinus and ligation of the jugular vein. Two others required secondary sinus operation.

c. Occluding thrombosis, seven cases. One patient, in whom free bleeding was obtained from below and without ligation, did splendidly, raising the question whether ligation is always necessary when free bleeding cannot be obtained from the bulbar end of the sinus. The mortality in this group alone was 25 per cent, and in the group as a whole, 24 per cent. The author is enthusiastic about immunotransfusions and the use of sera prepared by immunizing rabbits and injecting their serum into the patient. He also mentions the use of serum from a compatible donor instead of whole blood. In a small series of patients treated by this method, the mortality was only 15 per cent. He now reserves ligation as a secondary procedure, together with transfusion and revision of the wound, depending on the subsequent course of the disease. In his present series, ligation was undertaken in 10 patients, four of whom died. In the other six, the vein was collapsed in two instances, rendering it doubtful as to whether ligation was of any value. In the other four, ligation did not seem to be of appreciable benefit and the author questions the procedure's value unless there is thrombosis present in the vein itself.

This whole question of ligation or nonligation of the jugular vein in sinus thrombosis continues to occupy the attention of many investigators. The subject is comprehensively considered by Koch,[33] who feels that collections of statistics can never be entirely satisfactory and that the consideration of individual cases and the clinical indications for the particular method are, in general, more satisfactory. He lists the dangers of ligation as stressed by those opposed to the procedure as follows: *1.* Injury to the vagus nerve, which he feels is doubtful with competent technique. *2.* Edema in the region of the cerebrum, especially if the other jugular happens to be narrow. He has never observed this difficulty. *3.* Vascular stasis in the jugular and secondary retrograde stasis with propagation of infection. This objection, the author feels, is overcome by the fact that the sigmoid sinus must be opened to take care of the retrograde pressure and the eradication of the thrombus, if present. Free bleeding must be secured from above. If no visible thrombus is present, the upper end must be blocked off and the sinus opened. The jugular vein is the main channel and much more significant than the smaller tributaries, which usually close themselves off during the process of

thrombosis. The main thing, according to Koch, is to prevent access of infection to the larger vessels. With bulbar thrombosis, ligation and opening of the sinus is the only proper way to reach infection between the two points, in the mastoid and in the neck. By the author's technique, when the mastoid is opened and reveals any sinus or perisinus inflammation, the sinus is routinely uncovered and inspected. According to its condition, nothing more may be done. If there is free bleeding from the sinus, it is left alone. If it is thrombosed, it is first punctured, then widely opened, the thrombus removed, the cerebral end surely reached and broken down. The vein is then ligated. If the sinus is not opened, everything depends on the clinical course of the disease. If the temperature continues, if the patient's condition does not improve or if septic metastases appear, the jugular is ligated, the vein opened and blocked off cranially, even though the sinus may still contain fluid blood, lest a hidden and unapproachable thrombus be present. The author cites eight cases in which he feels definitely that jugular ligation was an important factor in the control of the infection and contributed definitely to the recovery in all instances. In seven years he has treated 34 cases by ligation, of whom nine died. This result he believes to be due to the late arrival of the patient and the presence of meningitis or metastatic sepsis in other organs. Fifteen other patients had severe thrombosis, in whom the jugular was opened and ligated and in all of whom recovery occurred. In 10 other patients there was no demonstrable thrombus but the sinus was opened and the jugular tied, with recovery. The author believes that in clinical otogenic sepsis, even without evidence of a thrombus, opening of the lateral sinus and ligation of the jugular vein is the best method.

Still pursuing the same subject, Atkinson[34] raises the question as to whether the jugular vein should be tied, merely because it has heretofore been customary and because operators claim to feel better if it is ligated. Anatomically, ligation does not exclude all parts whereby infection may spread, such, for instance, as the inferior petrosal sinus and the small veins passing through the posterior condyloid foramen. It is possible that the point of ligation may also serve as another focus if the process spreads, causing the very extension which one is trying to avoid. The author feels that the operation itself is at times much more difficult than one is led to believe

from much of the literature and that it may prolong and complicate the operation.

As an instance of spontaneous cure of sinus thrombosis, Atkinson cites the case of a girl, age 14 years, in whom, on a routine mastoid operation, a portion of the lateral sinus was represented by a fibrous band, apparently the result of previous infection with subsequent thrombosis, organization, sclerosis and spontaneous cure. He cites Koerner's figures in two series of presumably comparable cases in which the percentage of recovery was practically the same with ligation as without it.

In Greenberg's statistics of 61 cases there was a 15 per cent recovery in 21 instances of ligation, and a 60 per cent recovery in 40 instances of nonligation. In the first group, however, there was probably a larger number of extremely ill patients.

In the experimental work on dogs undertaken by Ungritz, the recovery with ligation and without ligation after production of experimental thrombosis was practically the same, and the experimenter concluded that ligation exercised no noticeable influence on the course of the disease.

Atkinson then undertook to operate on a number of patients without ligation by exposing the sinus widely at the pathological process and, in the absence of evidence of an occluding thrombus or a general infection, to do nothing more. If a definite thrombus was present or the sinus wall was necrotic or there were indications of a general infection, the sinus was opened and obliterated but the vein was not ligated. Only in the face of a general continuance of infection was operation on the neck undertaken. In the series of 15 patients, all recovered under his personal care. The patients were divided into three groups, as follows:

Group 1: Eight cases with pus in contact with the sinus wall. Treatment by full exposure but no interference with protective granulations and no opening or needling of the sinus. In only one instance was further treatment required. The author feels that some of these patients must have had a mural thrombus but apparently it was a protective mechanism and was left undisturbed, to continue its successful protection.

Group 2: Five patients with gross thrombus formation. The thrombus may be white, gelatinous and firmly attached to the wall of the vessel and easily removed in one piece, or it may be red, friable, loosely attached to the wall and liable to break in pieces during removal. Finally, the clot may have so broken down as to consist of frank pus actually in the lumen of the sinus. Treatment of this group consisted in removal of the upper end of the clot and from the lower end only so much of it as could be pulled out by gentle traction with forceps. No attempt was made to clear the bulb of any thrombus not readily removable in this manner.

Group 3: Two patients in whom there was necrosis of the sinus wall without thrombosis. Here, removal of the overlying bone may readily tear the sinus, with severe bleeding. The author believes ligation to be useless under these circumstances and that the prognosis is grave. He advocates ligation only if tenderness is evident over the course of the vein on gentle palpation. The presence of enlarged glands is significant and repeated chills after operation suggest a friable bulb clot. If more than two chills appear following operation, the author undertakes ligation and division of the vein with anchorage of the upper end to the skin surface.

In this connection, Sutherland[35] is authority for the opinion that ligation should be reserved, modified or omitted, according to the particular pathological conditions which are present. Clinically, he feels that three things are necessary in cases of sinus thrombosis: *1.* eradication and drainage of the primarily infected area; *2.* ligation of the internal jugular vein to prevent metastases and infiltration of septic material from the thrombosed sinus or vein into the general circulation; and *3.* the realization that walling off or resecting the vein may not be sufficient. It may be necessary to open the vein, evacuate its contents and allow for formation of granulation tissue. The complicated and complex anastomoses between the jugular above the point of ligation with the other craniovenous sinuses is well known. The author feels that the course of otitic sinus thrombosis may be greatly modified by anatomical anomalies in this venous system. The petrosquamous sinus is important in infancy and childhood before final obliteration occurs in the adult, and may play an important part in the formation of sinus complications. The question of liga-

tion is still a controversial one, but no single surgical procedure is adapted to all conditions.

In calling attention to the possibility that sinus thrombosis may be present in the absence of obvious otitis media, Childrey[36] cites the case of a man, age 28 years, in whom, despite absence of middle ear signs, mastoidectomy revealed a perisinus abscess. The sinus was not opened but the jugular vein was divided between silk ligatures. Temporary improvement was followed by discontinuance of sulfanilamide. Subsequently, the patient became irrational, complained of severe headache behind the right eye, and was subjected to exploration for a brain abscess, which was not found. The mastoid wound was reopened, the lateral sinus exposed and pus found to be coming from it. The sinus was widely opened, the thrombus removed and free bleeding obtained from the posterior limb. At autopsy a fresh thrombus was present in the remainder of the lateral sinus, extending across the torcula into the other side and also into the cavernous and petrosal sinuses. The petrous portion of the temporal bone was normal. The author raises the question of whether earlier opening of the lateral sinus would have changed the course of the disease, and whether ligation of the internal jugular vein was a factor in promoting dissemination of infection. The causative organism in this instance was a hemolytic staphylococcus, the clinical effects of which have long been known to be often more serious than in the case of the hemolytic streptococcus. The lack of definite symptoms of otitis media and the rapid progression of the infection to the cranial sinuses makes this case a most unusual one.

An interesting report is that of von Hoffmann,[37] of the case of a child, age 1 year, with an acute left otitis media and subperiosteal abscess of three weeks' duration. Following opening of the abscess and curettage of the wound, subsequent mastoidectomy was found necessary. High fever with septic remissions was unchanged by the administration of sulfanilamide. A striking feature was the appearance of marked stasis in all the veins of the skin of the head. Further revision of the mastoid wound revealed no evidence of sinus thrombosis. Suggestion of a right middle ear suppuration induced a mastoid operation on this side, with the finding of much pus under pressure and a marked sinus thrombosis with a fistula and pus within the vein. Bulb thrombosis was apparently evi-

dent and there was an extension of the venous stasis downward to the neck and chest, with final fatality. At autopsy there was found a thrombosis of both the transverse and sigmoid sinuses, the superior sagittal sinus, internal jugular vein and the right innominate vein, as well as the superior vena cava at its entrance into the right ventricle. Blood returning to the heart was thus forced to follow an external route via emissary veins and superficial vessels, such as the external jugular, explaining the dilatation of the scalp vessels. The obstruction to the jugular vein may have been aggravated by enlarged adjacent lymph glands.

The tendency to thrombosis in a child of this age is, in general, very uncommon. The chief clinical feature was a high, unremitting, septic temperature but without chills or local metastases. Strangely enough, the eye grounds showed no evidence of such extensive venous stasis. Interesting is the contrast between the infection in the two ears, the left with its more striking subperiosteal abscess, the right far less evident clinically but obviously harboring a much more virulent infection, leading the author to feel that evidence of a subperiosteal abscess with localization is always an indication of a favorable outcome.

Interesting is the importance assigned by Hargett[34] to complete immobilization and rest of the head in the treatment of sinus thrombosis. He cites the case of a man, age 37 years, who, following an injury to the nose, complained of earache and who developed in two weeks signs of acute mastoiditis. At simple mastoidectomy, the sinus was found exposed over a large area and covered with granulation tissue. Free bleeding was obtained upon opening the sinus, which was controlled with iodoform packs, removed on the fifth day. Subsequent pain over the site of the operation and fever of 102° were treated by 10 gr. doses of sulfanilamide three times a day. The head was elevated, heat was applied to the affected side and absolute rest was established. Subsequently, with permission for use of the bathroom, patient's temperature was immediately elevated but subsided on return to a regime of complete rest. This was continued for 23 days, when, with further trial of bathroom privileges there ensued an immediate temperature elevation to 102°. Further bed rest was prescribed for 45 days, when resumption of activities was uneventful. The author feels that sinus thrombosis is of some-

what the same nature as phlebitis in the leg, which, in the absence of high fever and chills, will respond to complete immobilization. On the same basis, the author stresses the value of immobilization of the head and absolute rest in bed. If, in spite of this treatment, there ensues a thrombosis with chills, higher fever, pain and systemic symptoms, further rest must be achieved by jugular ligation with removal of the overlying bony plates of the sinus to the limit of the thrombus formation. He feels that clots in the jugular bulb may be removed with a small rubber suction tube.

In discussing the general subject of sinus thrombosis, Friesner[39] and his co-workers call attention to the danger of overlooking pneumonia as a cause of the symptoms, likewise erysipelas. They do not advocate section of the jugular vein and feel that necessity for ligation must be decided by the course of the disease and the character of the symptoms rather than by any fixed rule for ligation. They believe sulfanilamide to be useful but think that its use should be avoided until the diagnosis is established, and that it then should constitute only an adjunct to surgical therapy. They endeavor to establish a blood level of about 15 mgm. per 100 cc., especially in the face of meningeal signs. Premature use of the drug may mask the clinical picture and, hence, the latter must always be evaluated with this fact in mind.

In an English symposium on sinus thrombosis, Colledge[40] calls attention to the fact that, whereas formerly sinus thrombosis was frequently associated with a chronic otitis media, more recently the percentage of this condition after acute otitis media has risen from 27 per cent 30 years ago to 71 per cent today. This situation, he feels, may be due to the lowered incidence of chronic otorrhea which, formerly so commonly neglected, is now avoided by such procedures as adenoidectomy and earlier mastoid surgery. In septicemia arising from thrombosis of the small mastoid vessels, he feels that operative treatment beyond complete removal of the primary focus of infection in the bone is not particularly effective. The author has no sympathy with the controversy between those who favor jugular ligation and those opposed to it, and quotes Barron as to indications for operation on the vein, as follows:

a. In acute pyemia and septicemia, whether the sinus is occupied by clot or by fluid blood.

b. If the sinus wall is gangrenous or its contents are liquefied, unless the area of infection be completely blocked off by noninfected thrombi at either end.

c. If it is proven or suspected that blood in the jugular bulb is wholly or partly clotted.

d. If the jugular vein itself is thrombosed.

Although for several years Barron has seen no fatal case in which the vein was not ligated, he does not feel that this proves that ligation is not beneficial and that dramatic results will not follow in properly selected cases. He believes that the ligated vein should be cut between ligatures and the upper end brought out of the incision in the neck. This technique has the added advantage of affording drainage to the bulb if indicated. Sulfanilamide, he believes, is very useful but too much cannot be expected of it unless a surgical focus is attacked.

Crowe[40] in his discussion was surprised to find in 800 mastoid operations only 1.3 per cent of sinus thrombosis, in contrast to 4 per cent commonly quoted in the literature. He suggests the possibility that by the time the idea of treatment for sinus thrombosis is discovered, there may be no more sinus thrombosis to treat. He prefers delayed intervention in simple mastoiditis and dislikes to operate under 10 days' time. In the 11 cases of sinus thrombosis under his care, there were two fatalities. He believes that jugular ligation frequently fails of its objectives and that dissemination of infection may occur in spite of this procedure through the collateral circulation. He believes the undisturbed clot is better protection than removal and ligation. This opinion appears to be substantiated by the great success which has followed the use of sulfanilamide. Future surgery for sinus thrombosis may comprise rapid excision of bone around a diseased sinus, gentle insertion of packing, which remains untouched for seven to 10 days, complete immobilization of the head and neck and the administration of sulfanilamide.

Layton,[40] in describing the pathology of sinus thrombosis, stresses the fact that there are no symptoms associated with a simple clot formation in the vein until it is invaded by an infecting organism. The first step, therefore, should be to remove the factory of organisms which are in contact with

the sinus wall. If symptoms then persist, one must suspect that the factory lies within the sinus, which should then be opened and any infected clot removed. Layton does not favor division of the vein in the neck or of bringing the end to the surface. During this same symposium, Diggle reported 13 cases of sinus thrombosis with four fatalities, of which meningitis was responsible for three. He feels that the sinus wall should be exposed in any suspicious case. If it looks and feels unhealthy, it should be left alone. If indurated and cord-like, it should be incised. In eight of nine patients, the jugular vein was ligated, but this was also true of four patients amongst whom there occurred three fatalities. He favors operation on the jugular vein for persistent fever and chills and in the absence of bleeding from the lower end of the sinus. He divides the vein between ligatures and feels that great additional benefit accrues from the use of sulfanilamide.

Somewhat at variance with these opinions is that of Kaplan,[40] who, at this same symposium, reported 400 cases of mastoiditis with no lateral sinus thrombosis. Of nine patients who died, all were operated upon after the eighth day, and amongst all the patients who were operated on before the eighth day there were no fatalities. This leads to the opinion that the frequently expressed idea that early operation is productive of complications may not rest on proved pathological findings.

Tremble[41] reports the case of a boy, age 5 years, who suffered from an acute right otitis media and in whom drum incision was followed by a profuse discharge for two weeks. Shortly after, the patient developed scarlet fever. Five weeks later, with the appearance of headache, elevation of temperature, granulations on the drums and mastoid tenderness, a right mastoidectomy was undertaken, the mastoid cavity being found filled with pus and granulation and cell partition being broken down into a large abscess cavity. The lateral sinus and middle fossa were not exposed, and the patient, after prompt improvement, was discharged from the hospital. Owing to persistent middle ear discharge after a month, however, re-exploration of the mastoid wound was undertaken and pus found exuding from underneath the temporal muscle. Temporary improvement was followed by a fever up to 106° and a leukocytosis of 17,000, for which situation the jugular vein was ligated. The wound was then exposed, the lateral

sinus examined and found to be healthy and on incision to present free bleeding. In spite of repeated transfusions, septic temperature persisted, and in the absence of evidence of meningitis the lateral sinus was re-explored from the torcula to the bulb and was found to be filled with a liver-colored clot, on removal of which free bleeding was established from above. The wound was treated with irrigations of acriflavine and with packing soaked in acriflavine. In spite of subsequent localized inflammation in the right hip and a pulmonary infarct, recovery ultimately followed after nine transfusions.

Here is a clear instance of the development of a thrombus in a vein which at the time of operation was clearly not thrombosed or apparently infected. The interesting question arises as to whether this subsequent thrombosis was in any way provoked by stasis incident to the early jugular ligation.

The unusual association between general purpura and acute otitic infection is recorded by Welk and Kasnetz.[42]. Their patient was a boy, age 7½ years, in whom the middle ear infection was followed by chills, fever and bleeding from the affected side, raising at first the question of phlebitis of the jugular bulb and its possible rupture. There was also evidence of nasal bleeding with clots in the nasopharynx. At simple mastoidectomy the lateral sinus was found thickened and discolored and covered with granulations and pus. In spite of transfusion, bleeding continued from the mastoid cavity and middle ear, likewise from the membranes of the nose and mouth. For seven days the patient received a daily transfusion. Following erysipelas, there next appeared subcutaneous hemorrhages in the shoulder, forearm, and the development of circumscribed abscesses, cultures of which showed a streptococcus viridans. The blood culture was positive for the next three weeks and the slowly healing mastoid wound was treated by packing the cavity with thromboplastin, by local heat, ultraviolet, quartz and mercury lamps. The sinus was never obliterated and the jugular vein was not ligated. The authors feel that in the majority of cases the diseased sinus wall, chills, bacteremia and typical irregular fever are not necessarily indications for jugular ligation. Splenectomy, though considered, was not carried out and repeated transfusions ultimately brought about recovery. An interesting question is whether the purpura was secondary to the otitis

media, mastoiditis or bacteremia, or whether it was an idiopathic condition complicated by an otitic infection.

An interesting article is that of Klicpera,[43] who discusses wounds of the lateral sinus. Operative injury to the lateral sinus may result from an anatomical anomaly, an abnormally thin sinus wall or an unusual location of the sinus, either so far forward as to hinder the operation or so superficial as to tend to injury the moment the cortex is opened. Pathological changes, such as sequestration in the neighborhood of the sinus, adhesions and attachments of the sinus wall to the surrounding bone, deposits of exudate, granulation or necrosis of the sinus wall are all factors which may lead the most experienced operator to an occasional accident. The wall may be injured with a chisel or sharp curette or by sharp slivers of bone or bony sequestra. Opening of the sinus wall may expose it to infection through direct bacterial invasion at the point of trauma, or in the presence of a perisinus abscess, infection may gain entrance through the diseased wall and thus produce a generalized blood stream infection. Moreover, the packing necessary to checking hemorrhage, especially if placed over a broad area, may induce local bacterial invasion with subsequent disease of the sinus wall and resultant secondary thrombosis. Fortunately, direct contact infection is usually warded off by the natural defensive powers of the body, particularly if there is only a single opportunity for access of bacteria. Moreover, the free bleeding usually present at the time of the injury tends to hinder such an invasion. The author feels that the best method of avoidance of subsequent infection after injury to the lateral sinus is a complete eradication of all infection in the mastoid cavities at the time of the injury, regardless of whether this has occurred in the middle of the operation or at its termination.

Work with experimental animals has demonstrated that in the presence of a bacteremia, even aseptic attacks on the sinus are almost always followed by the formation of infected granulations at the site of the sinus wound. Aside from local conditions, the state of the patient's resistance to the invading bacteria has much to do with the development of complications after injury to the sinus wall. An interesting question is that of possible danger of uncovering a presumably normal sinus wall. One author, in 300 exposures of the lateral sinus, reports six instances of secondary sinus thrombosis with a typical

septic course. In this author's statistics,[43] in 3,350 mastoid operations, there were 23 injuries of the sinus, of which three must be excluded as already showing signs of sinus sepsis before injury. Of the remaining 20, 17, or 85 per cent, ran a normal postoperative course. In the other three cases, injury was followed by a postoperative septic course, but at operation no sign of a thrombus was found in two instances. In the third instance, the thrombus appeared nine days after injury and required jugular ligation. In the 17 patients who ran a normal course after injury, the sinus was damaged in the process of seeking the antrum, and in four other instances injury was the result of alteration in the appearance of the sinus by granulations and bony necrosis. Injury arose both in the use of the chisel and in the curette and through the agency of bony splinters and sequestra. In only one instance did postoperative packing seem to produce any sign of stasis, such as headache, vomiting, etc. The author advocates as localized and isolating packing for bleeding from the sinus wound as possible. Its removal should be undertaken at least by the sixth day, though postoperative fever might indicate an earlier removal. The prime essential is the complete and thorough eradication of disease in the mastoid cavity at the time when the injury occurs.

PETROSITIS.

The subject of petrositis and the problems associated with its pathology and surgical treatment continue to occupy the attention of more and more otologists as this condition becomes more frequently recognized and as a better understanding is achieved of the appropriate methods of surgical approach to this formerly almost unexplored field.

Kopetzky[44] feels that it is usually possible so to locate the site of a lesion in the petrous pyramid that a specifically designed operation for its remedy can be executed. Of prime importance is the differentiation between a progressive and a recessive lesion. The former is indicated by sudden cessation of previously present pain and also by sudden cessation or marked diminution in the aural discharge, coupled with transitory photophobia, a late appearing abducens palsy and any one of a group of other symptoms indicating that the patient does not feel as well, is more irritable, wishes to be left alone and looks sicker than before. Such a picture indicates a pro-

gressive lesion even in the face of an afebrile reaction, absence of eye changes or decrease in the aural discharge. Surgical intervention is thus clearly indicated. Recession, on the other hand, is manifested by gradual diminution in the intensity and duration of the attack of pain with longer intervals and, likewise, with gradual diminution in the amount of discharge with lessened pulsation. If, in addition, the behavior and appearance of the patient indicate improvement, then spontaneous recovery of the petrous lesion may be expected. Direction of the type of surgical intervention depends on an accurate localization of the petrosal lesion. These may be posterior to the labyrinth or anterior. The former have a tendency to drain backwards into the mastoid cavity, whereas anteriorly lying lesions tend rather to drain into the tympanic cavity and the pus to find egress through the drum into the canal. If, after a complete simple mastoidectomy, there is excessive postaural discharge greater than one would normally expect in a healing wound, this is suggestion of a postlabyrinthine lesion. This situation indicates reopening of the wound and a search for fistulae in the exposed bony surfaces, either under the arch of the superior canal or along the prefacial mastoid cell tract leading toward the bulb. Treatment consists in the enlargement of these fistulae, curettement of their walls and drainage of the purulent accumulations from which they lead. Radical mastoidectomy, however, is in this postlabyrinthine type of lesion unnecessary. In the author's series of 10 such cases, all patients recovered. In the event of failure to locate such a fistula, radical mastoidectomy is a necessary step, particularly in the face of pulsating middle ear discharge which is pouring into the external canal. This situation indicates a lesion anterior to the cochlea, which is unapproachable by the simple mastoid operation. Only by the radical operation can subcochlear, supracochlear and precochlear lesions be approached, as well as closed empyemata in the pyramidal apex. The author has had 25 patients in whom tubotympanic fistulae were found, were enlarged and their contents successfully drained. In the event that no fistula is found, one must conclude the presence of an enclosed empyema in the pyramidal apex, a situation best relieved by the operation advised by Almour. Its advantage lies in the avoidance of bringing purulency in contact with the dura or other vital tissues. Obviously, in certain cases meningitis will

already have developed and rupture may have taken place into the posterior cranial fossa anterior to the cochlea, for which situation the best adapted procedure is that suggested by Lempert. The more extensive the lesion, the more radical must be the surgical attack. Kopetzky has operated upon 46 patients, with eight deaths. In 10 instances cure was obtained by simple mastoidectomy, in 25 by radical mastoidectomy, and in eight by operation according to the Almour technique, and in three by the technique of Lempert.

Kemler[45] believes that petrositis is more likely to be a complication of otitis media than of mastoiditis, its incidence being about one in 300 cases, especially in the presence of a well pneumatized mastoid process. The majority of instances of petrositis heal spontaneously but the recognition of those not likely to do so is important since, more and more, there is a growing feeling that patients dying of meningitis have suffered in reality from an unrecognized intermediary petrous infection. The author adopts the classification of Myerson and divides petrous lesions into four groups: *1.* apical empyema; *2.* osteomyelitis of the inferior aspect, a situation in which a collection of pus may break into the nasopharynx; *3.* granuloma, with or without pus formation, a form which has a very high mortality; and *4.* a vascular spread through the petrous pyramid, usually ending in fatality. In the author's series of six patients, three patients recovered without operation. He feels that the first indication is a thorough simple mastoidectomy, to be converted later into a radical if necessary, with wide exposure of the zygoma to facilitate examination of the orifice of the Eustachian tube and removal of the tensor tympani muscle. The technique of direct approach under the roof of the Eustachian orifice between the cochlea and the carotid canal has the great disadvantage of being hazardous except in the hands of a very few operators.

Myerson's operation of skeletonization of the superior semicircular canal may frequently reveal a softened patch of bone leading into the apex, from which pus can be liberated. If no such patch is found, the apex should be approached by way of the anterior pyramidal surface by elevation of the temporal lobe after adequate exposure. To facilitate this, removal of the spinal fluid may be carried out by lumbar or cistern puncture, thus lessening intracranial pressure. The squamous

portion of the temporal bone is removed to the size of a dollar. Useful landmarks as one progresses forward are the arcuate eminence, the subarcuate fossa, the roof of the internal auditory meatus and the roof of that portion of the petrous pyramid on which rests the Gasserian ganglion. By this approach a defect in the petrous wall will frequently be encountered, from which pus can be evacuated. Failing to find pus by this method, one may await its breaking through into the retropharynx.

An excellent summary of the subject of petrositis is a report of Moorhead[46] in a series of 30 consecutive cases of this condition with findings at operation of pus in the pyramid. In all but three instances there had been previous mastoid operations, varying from a week to several months before. In four cases the petrous infection followed a reinfection of an old healed mastoid with no previous signs or symptoms of petrositis. Moorehead calls attention to the fact that with an acute infectious process in the mastoid, one may have symptoms suggestive of petrositis without the necessity for any radical operative interference and with complete cure by the simple mastoid operation. Eye pain may not be typically in or behind the eye but may be around it or in the frontal region above it. In the author's series, only nine patients presented pain in the eye, one had no pain whatsoever, and one had pain in the eye on the opposite side. Pain in the cheek may be as significant as pain in or about the eye. Middle ear discharge is the most common finding, being sometimes profuse, but sometimes scanty. There is no characteristic temperature curve but all patients had some fever. In 14 cases there was an external rectus paralysis of varying extent. The author feels that X-ray is of value only as concerning clinical findings and is the least valuable and least reliable of all other signs. Stereoscopic films should be taken in the Stenver and Taylor positions. Transitory and incomplete facial paralysis was present in six of the author's cases and aided materially in the diagnosis. The later the onset of petrous symptoms after a simple mastoid operation, the more significant they are. External rectus paralysis following several days of pain and temperature signifies an extension of the suppurative process. Such paralysis invariably persists, whereas facial weakness may disappear. If no previous operation has been done, simple mastoidectomy alone should be done and the result awaited.

After a previous simple mastoidectomy, if the pain is not increasing, regardless of the discharge or its amount, the patient may be allowed to continue under close observation. The longer after simple mastoidectomy that the signs of petrosal involvement appear, the more the probability that further drainage will be necessary. The onset of these late symptoms warrants pyramidal exploration to forestall meningeal irritation. Any increase in the spinal fluid cell count would confirm this opinion. Moorehead raises the important question of the necessity of sacrificing hearing by the radical mastoid operation unless it is absolutely necessary. From his experience he feels that in the majority of instances the radical mastoid operation is unnecessary in order effectively to drain a focus of infection in the petrous pyramid. In the present series only one patient had a radical mastoidectomy, and that was done before he came under the author's care. In this series there were 10 deaths, but in eight of these the patients were operated upon in the presence of a fulminating meningitis and no other result could properly have been expected. The author uses a modification of Eagleton's operation of unlocking the petrous pyramid. The first step is a complete exenteration of the operative field by simple mastoidectomy. Where this has been previously done, the original incision is extended and the bone is removed to expose the dura of both the middle and posterior fossae. The bone between these exposures is then removed and the solid angle taken down between the posterior and middle fossa in order to reach the level of the labyrinth. This technique is much superior to simple removal of the bone overlying the dura of the middle fossa. Also, it permits tapping of the pontile cistern in those cases in which intracranial pressure is elevated because of meningeal irritation. With the author, tapping of the basal cistern is a routine procedure when the cell count in the spinal fluid is above normal or whenever it is desirable to reduce intracranial pressure. A search is then made for any tract leading into the pyramid and drainage is instituted along this area. If no tract is found, the dura of the middle fossa is gradually elevated and the anterior surface of the pyramid exposed. Pus may be encountered at this point, but if not, the cortex of the bone is opened with a curette or any other sharp instrument. Drainage is established by one or two strips of rubber dam and the wound left wide open. The chief objection to this

method is that it does not inspect the peritubal area, where there may be a peritubal fistula, though the latter, in the author's experience, is the least common site for petrous infection. The objection that too much dura is exposed by this method is not valid, nor is that which stresses too wide a decompression of the brain. The bone removed readily reforms, particularly in children. The objection that the dura is easily torn during elevation likewise has not, in the author's experience, been a deterrent and no case of meningitis followed such dural elevation. In fact, several of the patients recovered who had even had previous meningeal irritation, as shown by the condition of the spinal fluid. The percentage of recovery in this series is equal to that of any other author and no case was seen in which a radical mastoid operation could not be avoided.

In discussing this paper, Kopetzky quite logically pointed out that hearing after the usual radical mastoid operation may have already been greatly impaired because of long-standing chronic suppuration. A radical mastoid operation for petrositis in acute cases might not be found to impair the hearing anything like as much as one might expect as compared with the impairment resulting after radical surgery in chronic otitic suppuration.

In considering the question of petrositis and its surgical control, Carruthers[47] stresses the importance of regarding operative measures not only as a means of eradicating the local disease but also as a preventive against the development of meningitis, the earlier signs of which should always be looked for and recognized before it is too late. The problem is to know when to interfere further and when the advance inwards towards the meninges has or has not been checked. It is in this respect that the primary danger of inadequately or untreated petrositis lies. The proper attitude toward the development of meningitis should be that of not accepting it as an unavoidable termination in an unsatisfactory case but as a complication to be avoided by anticipating its development and directing proper surgical measures toward the site of its origin.

Page[48] pleads for the conservative treatment of petrositis in the majority of cases rather than the stressing of radical methods of operation in exceptional instances. Petrositis is

relatively rare after thorough mastoid surgery, the author having found only nine cases of petrositis in 3,465 cases of middle ear and mastoid suppuration at the Manhattan Eye and Ear Infirmary in five years. The variation in the number of reported cases of petrositis in different hospital clinics indicates a different diagnostic attitude by individual operators. Moreover, at postmortem in only one instance was meningitis found to be due to petrositis. The author believes that simple procedures should be tried first before progressing to more radical ones. Every simple mastoidectomy should be as complete as the operator can make it to prevent further development of complication in deeper, unopened cells. The same attitude used to be taken toward labyrinthitis which followed incomplete simple mastoid operation and which is now slowly disappearing. Labyrinthine operations were formerly performed without adequate indications, and the same may now be true of petrositis.

If, after simple mastoidectomy, pain or headache appear, then secondary operation is indicated with widening of the aditus and the removal of cells near the superior semicircular canal. Attention is also indicated to cells deep in the base of the pyramid behind and internal to the facial nerve and between the posterior semicircular canal and the jugular bulb. If a wide open wound here does not give relief, then radical mastoidectomy should be considered. Only after that should a radical exploration of the apex be undertaken, and only in exceptional cases will such drastic measures be required.

In a discussion of the principal sites of cellular infection in the petrous pyramid, Tobeck[49] stresses the relationship of these infections to the middle ear. Beginning with an occlusion of the opening of the pyramidal cells and hence an obstruction to the drainage of pus forming in them, there occurs subsequent bony destruction of the cell walls and irritation of the adjacent nerves, particularly the trigeminus and abducens. The great danger is that of meningitis because of adjacent dural infection and a breaking through into the carotid canal with septic thrombosis of the carotid venous plexus and other venous sinuses. Although infection may break into the semicircular canal, the labyrinth and cochlea, this type of extension is rarer, as is also brain abscess. The author believes that 50 per cent of the cases of fatal otitic meningitis are

associated with some form of pyramidal infection. Meningeal symptoms may come suddenly and measures for their treatment may be applied too late. Petrous symptoms may be present at the onset in otitis media or may come on later, in from four to six weeks. The chief thing of importance is to suspect the possibility of petrositis as an explanation for untoward postoperative symptoms after simple mastoidectomy. Repeated lumbar punctures are an important diagnostic feature, and a study of the cellular characteristics of the spinal fluid will yield valuable information. Even with adequate surgical drainage, there will be certain foci in the petrous pyramid which will result in meningitis because they cannot be completely reached surgically. Operations may conveniently be divided into those outside the pyramid, those inside the pyramid, with drainage of a focus, and finally operation, in which exposure of the focus necessitates destruction of the labyrinth. It must be recognized that, although the operations themselves frequently entail considerable risk, the lesions for which they are applied are so serious as to justify their execution.

Gullermin[50] and his co-workers feel that, in spite of the classical picture of petrositis so frequently quoted, one must be on guard against a latent form of this disease, and cites a case in which a patient with some signs of external rectus paralysis and facial paralysis, but with absolutely no pain or aural discharge, was operated upon. Twelve days after operation, patient died of meningitis, and postmortem examination showed an extensive lesion in the petrous pyramid.

In a discussion of operative procedures for the treatment of petrositis, Jones[51] advocates the approach used by many neurological surgeons for the Gasserian ganglion. This technique, he feels, gives complete visualization of the superior surface of the petrous pyramid. It is undertaken through a sterile field, avoids elevation of the dura from an infective mastoid process, hence does not run the risk of a complicating meningitis through accidental laceration of the dura. Patient is in the sitting posture to reduce intracranial pressure, thus rendering dural retraction easy. With preliminary sedation, local or general anesthesia may be used. A perpendicular incision extends from the attachment of the temporal muscle downwards to the zygomatic process and then anteriorly to

the external meatus. Following elevation of the periosteum to give as wide an exposure as possible, the trephine opening is made with a Hudson burr and enlarged with rongeurs. The dura is next elevated around the floor of the middle fossa, great care being used where it is adherent. At the location of the middle meningeal artery, the latter is occluded by a peg inserted in the foramen spinosum. Severing the artery permits further elevation of the dura and exposure of the third division of the Vth nerve as it passes through the foramen ovale. This procedure exposes the bony roof of the petrous pyramid containing the major cellular elements. The carotid artery may have to be depressed in order to permit complete removal of the cells of this portion. Drainage may be carried out posteriorly through the mastoid wound or laterally through the avenue of approach. Bleeding from the carotid artery may possibly require ligation. This procedure is suitable for reaching the following types of infection: *1.* extradural abscess in the middle fossa, resulting from exit of pus from the petrous pyramid; *2.* extradural abscess from the posterior fossa, of similar origin; *3.* empyema of the antelabyrinthine portion of the petrous pyramid; *4.* pericarotid abscess; and *5.* in the operative attack on cavernous sinus thrombosis.

In studying the pathology of petrositis, Lindsay[52] found that the predominating pathological lesion was a coalescent osteitis within the pneumatic spaces, producing destruction and gradual erosion of the cortex, either directly or along vascular channels in the bone to the middle or posterior cranial fossa, or both. In those instances in which there was also present a diffuse meningitis, there was evidence that the suppuration had remained localized for a considerable period of time and, hence, might have been then amenable to surgery. In addition, there was marked evidence of a proliferative process in the bone marrow, with resultant fibrosis and in some areas sclerosis, providing a line of defense against invasion of the marrow spaces by suppuration in the adjacent areas of bone. In only one instance was there found an acute osteomyelitis of the petrous pyramid. In 11 cases the hemolytic streptococcus was found responsible in seven, type III pneumococcus in three, and type I pneumococcus in one. Jugular thrombosis may be associated with apical suppuration, but unless the latter is independently cared for, ligation and exci-

sion of the jugular will not prevent meningeal extension. No single operative procedure is suitable for all types of petrositis. The first essential is complete exenteration of the mastoid cells with exposure of the dura of the middle and posterior fossae. If the labyrinth functions normally and no fistulous tract is evident, the dura must be elevated beyond the arcuate eminence in the middle fossa. Apical abscesses are, in general, not accessible from the mastoid. While a fistula into the middle ear may permit spontaneous recovery or act as a lead to the focus in the apex, the commonest situation is that of a closed abscess. The author's procedure was adoption of a radical mastoid operation with extension by removal of the processus tympanicus, the zygomatic root, the anterior canal wall and the tensor tympani muscle. The apex was then entered from in front of the cochlea by means of a Eustachian tube curette. The chief risk of the intrapetrosal approach from the middle ear is hemorrhage, but it is the most practical for closed abscess of the apex. One must recognize that local meningeal inflammation may be present before changes are evident in the spinal fluid. Pain or headache was the most reliable constant symptom. The author has found X-ray most helpful in diagnosis, particularly in Stenver's position, but it gives no information as to when or where intracranial extension is likely to take place.

In a splendidly written and meticulously detailed article, Wishart[53] reports three instructive cases of different types of petrosal infection, drawing from them certain important conclusions. The first is that of a girl, age 12 years, with longstanding history of recurrent cervical adenitis, who finally died of meningitis, autopsy revealing a large abscess in the neck, which had broken through into the petrous pyramid. There was a possibility of an underlying tuberculous condition. The second case was that of a boy, age 12 years, with a typical symptom-complex of petrositis with classical symptoms. After a series of operations progressing from simple to a radical mastoidectomy, spontaneous drainage of the infection in the petrous pyramid took place and the patient gradually recovered. In the third case, a girl, age 17 years, developed an acute middle ear infection with mastoiditis treated by simple mastoidectomy. Meningitis gradually developed, a radical operation was done and every effort made to find a fistulous lead near the Eustachian tube, without success.

Patient gradually became worse and finally died of what was suspected to be a cerebellar abscess. Autopsy, however, revealed a large abscess in the petrous tip.

Wishart intimates that possibly the operation by the Kopetzky and Almour technique might have avoided this tragedy in spite of the risk entailed in its execution. He then goes on to trace the rise of importance of the subject of petrositis from the time when no mention of it could be found in the literature. He discusses the present concept of the structure of the petrous bone, consisting both of pneumatic cells and of marrow cells, with the utmost variation in the extent and relationship of these two types.

There is still no agreement as to the points of origin of the pneumatized petrous cells, whether from a single area near the orifice of the Eustachian tube or from multiple areas, as claimed by Guild. This situation has definite bearing on the surgical approach since to curette and break down infected pneumatic areas is good surgery, whereas the same approach to infectious osteomyelitic areas is not. Air cells become infected through their point of origin, whereas marrow cells may become infected either from adjacent air-containing cells or by way of the general blood stream, as seen in osteomyelitis of the long bones. Thus, empyema of the petrous tip raises the problem of whether the surgeon shall approach it from outside the petrous bone, through the petrous bone or merely by exposure of the Eustachian tube area. The approach from without, by way of the middle fossa, can be safely made only to within a few millimetres of the Gasserian ganglion. Attack beyond this point risks rupture of the arachnoid with almost inevitable meningitis. Only the fortunate encounter of an extradural abscess with the ability to probe or curette the petrous tip through a walled-off area can justify this approach. The posterior cranial fossa approach is even more hazardous, though the complicated and time-consuming technique of Lempert is an attempt to solve this problem. The intrapetrosal approaches of Ramadier and of Almour both have serious objections because of difficulties and risk. It is the author's opinion that the present vogue for petrositis has given it a prominence and appearance of frequent occurrence not justified by a large group of patients. In 15 years, in 3,425 consecutive autopsies at the Hospital for Sick Children,

in Toronto, suppuration of the petrous tip was discovered in only one case. In the Ear, Nose and Throat Department of the Royal Infirmary, in Edinburgh, in 1,279 consecutive cases of mastoiditis which came to operation, including all those showing intracranial complications and which came to post-mortem, there was only one case of meningitis following apicitis.

In attempting to explain this situation, Wishart doubts that it is the result of failure to discover petrositis at autopsy and feels that, more likely, petrositis may have been present in some cases of intracranial complication which recovered. He feels that most patients with petrositis will recover with a thorough, properly done mastoid operation and with proper after-care. The Gradenigo syndrome with VIth nerve paralysis is by no means assurance of the presence of petrositis. X-ray is helpful but is less important in an uncertain and suspected case than in one in which definite clinical symptoms suggest petrositis. It is more important when negative than positive, since many cases showing X-ray changes have recovered with conservative treatment. The author suggests that X-ray study of the petrous be confined to cases where the clinical studies suggest the possibility of suppuration. He feels that an examination of any large number of cases of otitic sepsis justifies the opinion that the majority of suppurations in the petrous pyramid will drain and heal spontaneously.

BIBLIOGRAPHY.

1. Dwyer, J. G.: Surgical Indications in Suppurative Meningitis. The Laryngoscope, 48:7:461-471, July, 1938.

2. Friesner, I., and Rosenwasser, H.: Surgical Treatment of Meningitis. The Laryngoscope, 48:7:472-479, July, 1938.

3. Kopetzky, S. J.: The Diagnosis and Therapy of Otogenic Meningitis. *Pa. Med. Jour.*, 42:3:217-225, Dec., 1938.

4. Yaskin, J. C.: Differential Diagnosis of Bacterial Meningitis of Aural and of Nasal Origin. *Arch. Otolaryngol.*, 27:4:444, April, 1938.

5. Garfin, S. W.: Meningitis (Otitic) Due to Type III Pneumococcus; Operation — Recovery. The Laryngoscope, 48:2:793-801.

6. Jackson, C. C. Rae: Petrositis, Meningitis, Sulfanilamide — Drainage — Recovery. The Laryngoscope, 48:9:689-692, Sept., 1938.

7. Cline, Samuel: Streptococcic Meningitis. *Med. Rec.*, 147:334, April 20, 1938.

8. Halphen, Martin, and Bignotti, R.: Meningite a streptocoques, d'origine mastoidienne, etc. *Les Annal. d'Oto-Laryngol.*, 11:1033, Nov., 1938.

9. HUBERT, C.: Quatre cas de meningite post-otique. *Rev. de Laryngol., Otol. and Rhinol.*, 59:4:365, April, 1938.

10. SMITH, F. W., ET AL.: Streptococcus Meningitis of Otitic Origin — Treated with Sulfanilamide — Recovery. *Jour. Amer. Med. Assn.*, 110:12:887, March 19, 1938.

11. ROBERTS, E. R.: Bacterial Otitic Meningitis. *Jour. Conn. State Med. Soc.*, 2:4:185, April, 1938.

12. LAW, T. B.: Otogenous Meningitis with Recovery. *Med. Jour. Australia*, 1:10:448, March, 1938.

13. MARTIN, H. I., and ELLENBERG, S. L.: Beta-Hemolytic Streptococcus Meningitis. *Arch. Pediat.*, 55:428, July, 1938.

14. GORDON, C. H.: Case of Mastoiditis and Influenzal Meningitis — Recovery. *Me. Med. Jour.*, 29:4:66, April, 1938.

15. SACKS, PHILIP: Hemolytic Streptococcus Meningitis of Otitic Origin — Report of a Recovery. *Arch. Otolaryngol.*, 28:3:364, Sept., 1938.

16. APPELBAUM, E.: Sulfanilamide in Treatment of Meningitis, etc. THE LARYNGOSCOPE, 48:7:482-498, July, 1938.

17. DIEHL, J. E.: Meningitis Secondary to Otitic Infection. *Va. Med. Mon.*, 65:750-752, Dec., 1938.

18. SMITH, A. B., and SCOTT, C. E.: Streptococcal Mastoiditis and Meningitis Following Tonsillectomy. *Jour. Laryngol. and Otol.*, 53:4:256, April, 1938.

19. ADOLPH, P. E., and LOCKWOOD, J. S.: Studies in the Mechanism of the Action of Sulfanilamide. *Arch. Otolaryngol.*, 27:5:535, May, 1938.

20. CINELLI, A. A.: Clinical Forms of Labyrinthitis. THE LARYNGOSCOPE, 48:3:157-167, March, 1938.

21. WAILS, T. G. Clinical Labyrinthitis. *Jour. Okla. S. M. A.*, 31:6-9, Jan., 1938.

22. BRADBEER, W. H.: Case of Acute Labyrinthitis. *Jour. Laryngol. and Otol.*, 53:2, Feb., 1938.

23. SERCER, A.: Bericht uber die an der Ohrenklinik in Agram beobachteten Gehirnabszesse. *Monatsschr. f. Ohrenheilk. u. Laryngo-Rhinol.*, 72:10:946-958, Oct., 1939.

24. MEURMAN, Y.: Zur Klinik und Pathologie der Otogenen und rhinogen Hirnabszesse. *Acta oto-laryngol.*, 26:5:451, 1938.

25. LASKIEWICZ, A.: La lipiodographie des abces cerebraux. *Rev. de Laryngol., Otol., Rhinol.*, 59:3:217, March, 1938.

26. BRUNNER, H., and DINOLT, R.: Uber die otogene Encephalitis purulenta progressive. *Ztschr. f. d. ges. Neurol. u. Psych.*, 162:106-125, 1938 (need of differentiating this from brain abscess).

27. BRUNNER, H., and DINOLT, R.: Zur Pathogenese und Pathologie des otogene gross Nirnabszesses. *Monatsschr. f. Ohrenheilk. u. Laryngo-Rhinol.*, 72:3:255-276, March, 1938.

28. TROMEUR, E.: Abces latent du cerveau. *Les Annal. d'Oto-laryngol.*, 12:1186, Dec., 1938.

29. BACY, P. C.: Sulfanilamide in Treatment of Brain Abscess. *Jour. Amer. Med. Assn.*, 3:14:1639, Oct. 29, 1938.

30. GUNS: Discussion therapeutique de l'abces du cerveau. *Les Annal. d'Oto-laryngol.*, 1:41, Jan., 1938.

31. Evans, W. H.: Thrombosis of the Lateral Sinus. *Arch. Otolaryngol.*, 28:6:959, Dec., 1938.

32. Juers, A. L.: Otitic Sepsis. *Arch. Otolaryngol.*, 27:2:178.

33. Koch, F. X.: Beiträge zur Jugular is Unterbinding bei Sinus Erkrankungen am Mittelohr. *Monatsschr. f. Ohrenheilk. u. Laryngo-Rhinol.*, 72:7:672-681, July, 1938.

34. Atkinson, E. M.: Treatment of Lateral Sinus Infections Without Operation on the Jugular Vein. *Arch. Otolaryngol.*, 27:2:169, Feb., 1938.

35. Sutherland, J. M.: Otitic Sinus Thrombosis. *Arch. Otolaryngol.*, 27:1:1, Jan., 1938.

36. Childrey, J. H.: Sinus Thrombosis and Mastoiditis Without Otorrhea. *S.W. Med.*, 22:5:182, May, 1938.

37. Hoffmann, L. von: Otogene Thrombose beider Sinus transversi, etc. *Monatsschr. f. Ohrenheilk. u. Laryngo-Rhinol.*, 72:12:1173-1178, Dec., 1938.

38. Hargett, E. R.: Rationale and Treatment of Sigmoid Sinus Inflammation. The Laryngoscope, 48:2:126-128, Feb., 1938.

39. Friesner, I.; Druss, J. G.; Goldman, J. L., and Rosenwasser, H.: Sinus Thrombosis. *Amer. Jour. Surg.*, 42:116, Oct., 1938.

40. Discussion on Lateral Sinus Thrombosis. Society Proceedings, Royal Society of Medicine, Section on Otology. *Jour. Laryngol. and Otol.*, 53:8, Aug., 1938.

41. Tremble, G. E.: Scarlet Fever Mastoiditis with Sinus Thrombosis. *Canad. Med. Assn. Jour.*, 38:4:378, April, 1938.

42. Welk, B., and Kasnetz, J.: Thrombopenic Purpura as a Complication (of Otitic Sepsis). *Arch. Otolaryngol.*, 27:6:732, June, 1938.

43. Klicpera, L.: Uber operative Verletzungen des Sinus sigmoideus und ihre Folgen. *Monatsschr. f. Ohrenheilk. u. Laryngo-Rhinol.*, 72:11:1059-1066, Nov., 1938.

44. Kopetzky, S. J.: Surgical Treatment of Various Types of Lesions in the Petrosal Pyramid. *Surg., Gynecol. and Obstet.*, 66:2A:395, Feb., 1938.

45. Kemler, J. L.: Clinical Manifestation of Petrositis and Its Treatment. *South. Med. Jour.*, 31:12:1261, Dec., 1938.

46. Moorhead, R. L., and Baker, J. P.: Suppuration of the Petrous Pyramid. *Arch. Otolaryngol.*, 27:4:497.

47. Carruthers, D. G.: Review of Problems of Suppurative Petrositis, etc. *Med. Jour. Australia*, 2:16:644, Oct., 1938.

48. Page, J. R.: Conservative Steps in the Treatment of Petrositis. *Jour. Conn. State Med. Soc.*, 2:6:275, June, 1938.

49. Tobeck, H.: Die Pyramidenzellen Eiterungen. *Med. Klin.*, 34:24:797-799, June, 1938.

50. Gullermin, M.; Pesme, J., and Condane, R.: Petrosite latente. *Les Ann. d'oto-laryngol.*, 8:729, Aug., 1938.

51. Jones, M. F.: Modifications of the Semilunar Ganglion Approach in Petrous Pyramid Surgery. The Laryngoscope, 48:10:738-744, Oct., 1938.

52. Lindsay, J. R.: Suppuration in the Petrous Pyramid. *Annal. Oto-Rhino-Laryngol.*, 47:1, March, 1938.

53. Wishart, D. E. S.: Some Observations on Petrous Tip Suppuration. *Canad. Med. Assn. Jour.*, 39:1:50, July, 1938.

54. Kopetzky, S. J.: Management and Treatment of Otitic Meningitis. *Annal. Oto-Rhino-Laryngol.*, 47:117-134, March, 1938.

THE PROGRESS OF DEAFNESS IN CLINICAL OTOSCLEROSIS.*†

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There is quite general agreement among authorities as to the progressive nature of the deafness in cases of clinical otosclerosis. Lederer[1] in his new text says, "The deafness begins unilaterally and is insidious in its onset. . . . Eventually both ears become affected, resulting in a high degree of deafness. . . . Increasing deafness, coming on for months and even years, should arouse suspicion of this disease entity." Kerrison[2] says, "The impairment of hearing is frequently so gradual in its advance as to be fairly pronounced before the patient is fully aware of his functional loss. In the early stages the impairment of hearing may be manifested by slight loss of auditory acuteness for the watch, acoumeter and the conversational voice, and by very slight increase in hearing by bone conduction. . . . The course of the disease varies greatly in different cases. In some cases the disease progresses rapidly from the start, leading within a comparatively short period to very marked — sometimes to profound — deafness. . . . Fortunately, there is a large class of cases in which the disease is very gradual in its advance. In some cases years may elapse without apparent progress in the lesion." Holmgren,[3] after an interval of from 11.2 to 17.2 years, re-examined the hearing of 66 of his otosclerotic patients and found that in 16 the exacerbation was marked, in seven it was marked and rapid, in 21 it was moderate, in two it was slight, in seven it was slight and slow, in six there had been no progress, and the progress in seven could not be determined because the deafness was so marked at the first test. He states, "The patient's hearing fluctuates, depending upon many various circumstances. If the patient is in good condition, the distance becomes greater, and if he is in a poor condition (as a result of loss of sleep, nervousness, the influence of sedatives, etc.), the distance becomes shorter. Psychic

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conditions play an important part. If the patient 'wills to hear,' the distance becomes greater. If the patient is tired, depressed or indifferent, the distance becomes less. . . . All of this is well known to the experienced otologist. I mention it

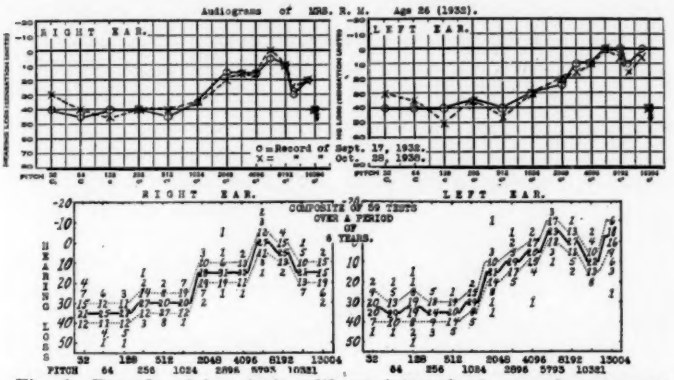

Fig. 1. Records of two tests with an interval of over six years are shown at the top. Fifty-nine tests of this patient are tabulated at the bottom. The heavy broken line passes through the thresholds most frequently recorded. The parallel lines of dots are drawn 5 units above and below this heavy line.

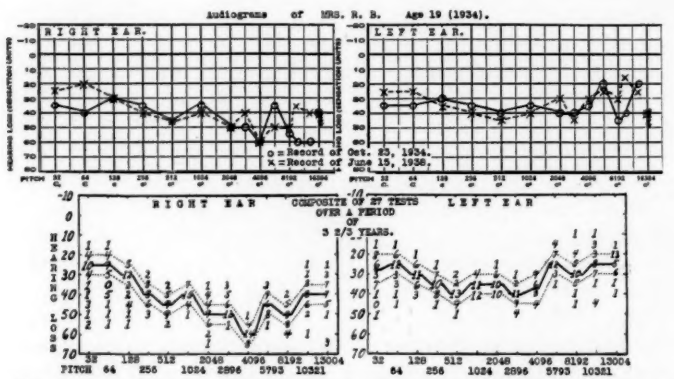

Fig. 2. Records of a patient who has been tested 27 times during a period of three and one-half years, treated similarly to those shown in Fig. 1.

here in order to emphasize the fact that the whispering distance and conversational distance, even if recorded by the most experienced and trained examiner and under the most favorable conditions, have only a limited value and must under no

consideration be taken as a sure gauge of the patient's hearing and as such used for comparison with values obtained at another examination. . . . The best method for obtaining objective and commensurate values is to make use of an audiometer. Hearing results following operations for otosclerosis recorded only by whispering and conversation distance are not reliable, provided the differences and distances are not great."

If, then, otosclerosis be progressive as defined and described by these authorities, just what prognosis shall the otologist give his otosclerotic patient? Shall he tell the patient that his

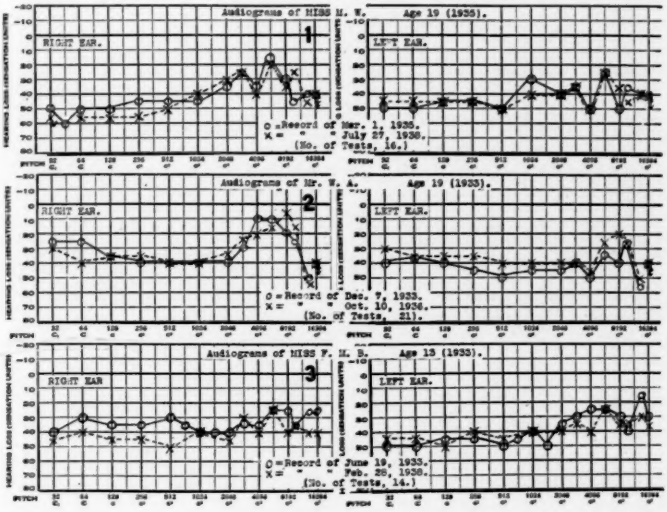

Fig. 3. First and last records of three patients who have been tested repeatedly during periods ranging from two and five-sixth years to four and two-third years. All were under age 20 years at the time of the first test.

deafness will become more marked? If so, will the progress be slow or rapid? How long will it be before the patient will cease to have useful hearing? Are there any conditions which tend to aggravate the deafness? These are some of the questions which have brought about this study.

It appears that these problems may be solved by either of two methods: first, by testing the hearing of a large number of patients throughout life and carefully studying changes as

they appear; or, second, by testing a large number of cases at different age levels and by statistical methods to determine the amount of change at each age level. This report covers the attempts which have been made, using both these methods, in a very small way during the last 10 years in an attempt to secure some hint as to the answers to these questions.

It should be stated that the diagnosis of otosclerosis in each of the cases presented was made by competent otologists on the staff of the Department of Otolaryngology at Washington University. The classical picture secured by the fork tests, together with the absence of tubal or tympanic pathology, was

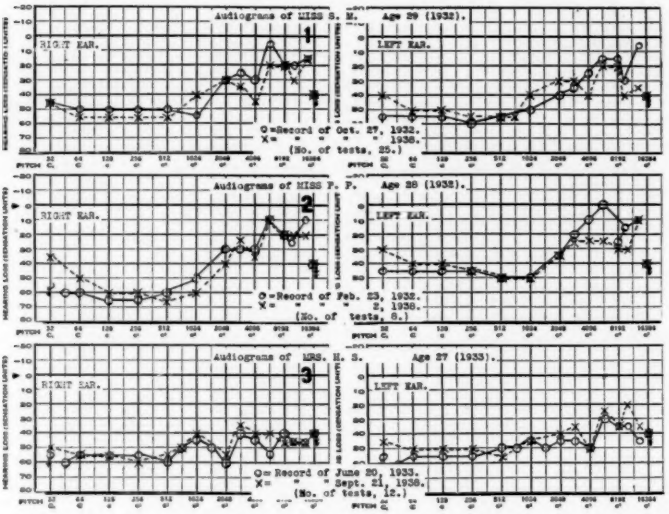

Fig. 4. First and last records of three patients who have been tested at intervals during a period ranging from five and one-third years to six years. All were within the third decade at the time of the first tests.

the basis for diagnosis. All audiometric tests were done by the writer, using a Western Electric 1A Audiometer. In no case was the diagnosis made on the basis of the audiometric tests alone.

One of the first problems which arises in any audiometric study is to determine the accuracy of the patient's response. That there must be variations in any two tests of the same patient is as true of audiometric tests as it is of any func-

tional test. How great shall this variation be before it shall be considered significant? Suggit[4] says, "No treatment of otosclerosis can be considered of value unless it can produce a sustained improvement in hearing appreciably greater than 10 dcb. over the greater part of the range, 64 to 8,192 d.v." The writer interprets this to mean that changes less than 10 dcb. are indefinite. This variable, which might be called the patient's attention factor, is illustrated in Fig. 1. At the top of the chart are the audiograms for the right and left ears secured in two tests, taken approximately six years

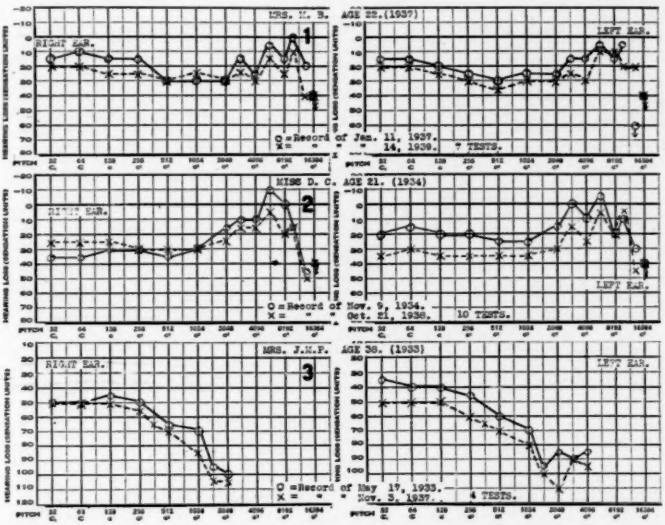

Fig. 5. First and last records of three patients who have been tested at intervals from two years to four and one-half years which show a questionable progress of the deafness.

apart. The records for the right ear show that seven tones have the same threshold in both tests, six had a variation of 5 dcb., and for one tone the variation was 10 dcb. The left ear shows the same threshold for five tones, seven tones varied 5 dcb., and two tones showed 10 dcb. variation. More than this, in each ear the curves cross several times. This is as close to identical records as the writer has been able to obtain in repeated tests of the same patient, no matter what the time interval may be. This patient, diagnosed as having otosclero-

sis, was tested 59 times during the six years. The thresholds of the 59 tests are tabulated as a scattergram at the bottom of the same figure, which shows considerable variation in the threshold readings. For example, in the right ear the tone of 1,024 d.v. was heard seven times at 20 dcb., 19 times at 25 dcb., 20 times at 30 dcb., 12 times at 35 dcb., and once at 40 dcb., the scattering being from 20 dcb. to 40 dcb. This may be considered a wide variation; however, if a line be drawn through those points where the greatest number of thresholds are recorded (illustrated by the heavy broken line in the

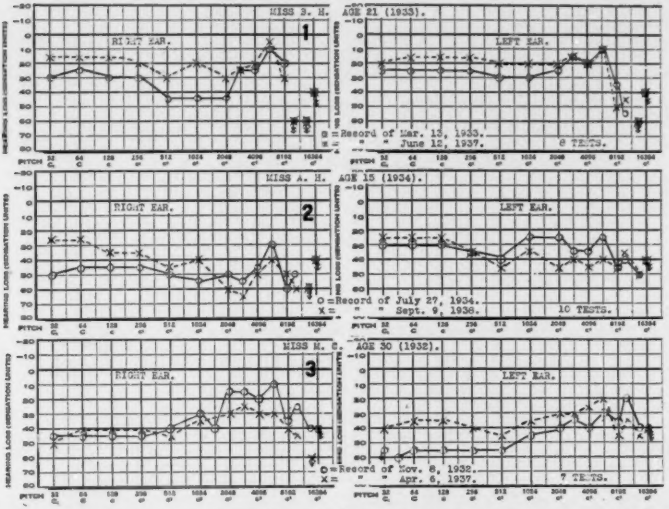

Fig. 6. First and last records of patients who have been tested at intervals for more than four years. These records show questionable improvement in hearing in one or both ears.

charts), and parallel lines drawn at a distance of 5 dcb. from this broken line (illustrated by the two lines of dots), the area included between the dotted lines, which represents a plus or minus variation of 5 dcb., contains approximately 80 per cent of the entire number of threshold readings. The records for the left ear show a similar variation, except that at 32 d.v. and 128 d.v. the variation is 7.5 dcb.

Fig. 2 shows a similar treatment of the records of another otosclerotic patient who has been tested 27 times during a period of three and two-third years. In Fig. 2 the scattering

appears greater, but it must be remembered that fewer tests are recorded. Neither of these patients was aware of any increase in deafness while she had been under observation. On the basis of these two charts, in this study at least, changes greater than plus or minus 5 dcb., or 7.5 dcb. at the most, must be considered significantly greater than the attention factor.

Fig. 1 reveals other pertinent information. The close approximation of the records secured during this period of

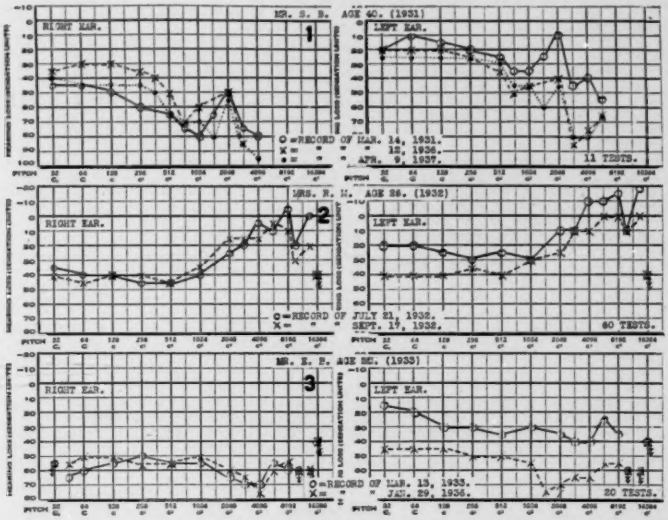

Fig. 7. Records of three cases which show definite progress of the deafness. In No. 2 the loss in the left ear was evident in three months.

six years indicates that no measurable change in the calibration or output of the audiometer is apparent. If changes have taken place, they are less than the listener is able to determine or record. The instrument has been in constant daily use for over 10 years. The necessity for constancy of technique and stability of calibration in a study of this character is apparent.

Fig. 3 shows the records of three patients, all under age 20 years at the time of the first test. For convenience in comparison, the records of the first and last tests for each ear

have been plotted on the same chart. No. 1 has been tested 16 times in three and five-twelfth years; No. 2, 21 times in two and five-sixth years; and No. 3, 14 times in four and two-third years. In each chart the curves of the two records cross several times, and only in the right ear of No. 3 according to the criteria above can there be said to be an increase in the deafness, and in this ear the additional loss is for tones below 1,024 d.v. The loss is 20 dcb. at 512 d.v. but is otherwise within the limits of observation. By the same standard, the records for the left ear of No. 2 indicate improvement, but the amount of gain is not greater than 10 dcb. for any tone and is probably within the limits of observational error. All

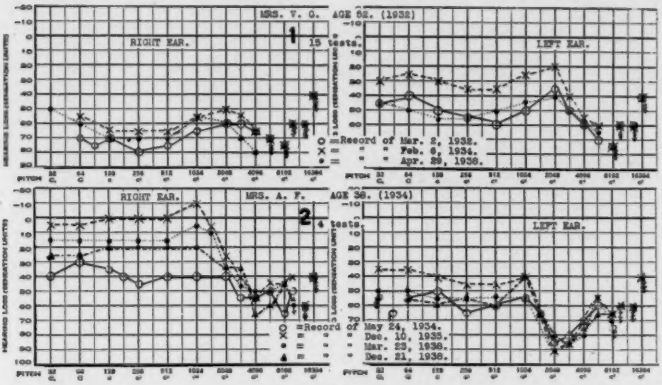

Fig. 8. Records of two cases showing definite improvement in hearing.

show losses of 50 dcb. or less. (The normal curve for tests in a soundproof room has not been drawn in these charts because it is not essential for comparison.) It is concluded from the six records that four show no change, and that the changes in the other two are questionably beyond the limits of observational error.

This is illustrated again in Fig. 4. All of these patients were in their twenties at the time of the first test. No. 1 has been tested 25 times in six years, No. 2 eight times in six years, and No. 3 12 times in five and one-third years. The manner in which the curves cross in each record indicates that no measurable increase in the deafness has taken place.

A comparison of the records in Figs. 4 and 5 shows about the same degree of loss, regardless of the fact that the patients whose records are shown in Fig. 4 are 10 years older than those shown in Fig. 3. While the records are not entirely similar in contour, all show about 40 to 60 dcb. loss through the speech range; *i.e.*, from 300 to 3,000 d.v.

Fig. 5 shows the records of three patients with a questionable slight increase in hearing loss. In the left ear of No. 1 almost every tone is lower in the last test than in the first

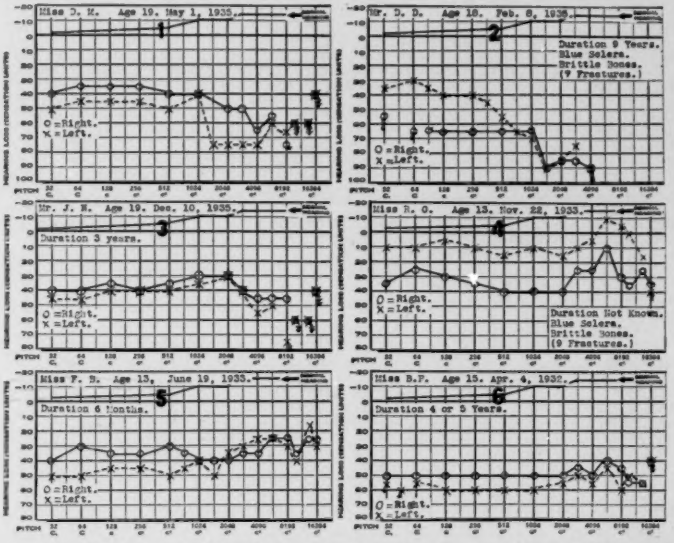

Fig. 9. Records of six patients under age 20 years. No. 2 shows the most marked hearing loss.

by 5 dcb., yet because the curves do not cross, the loss may be definite. The left ear of No. 2 shows a loss of from 10 to 15 dcb., while the right shows no progress. Both ears in No. 3 show from 10 to 15 dcb. increase in the deafness.

By the same standard, the records shown in Fig. 6 indicate questionable improvement. Both ears of No. 1, in tests approximately four years apart, give better records in the most recent test. This patient, a nurse, was told by her otologist that she would eventually become deaf and that she

should learn lip-reading at once. She became mentally upset when this prognosis was given, but as she watched the results of the tests from time to time she "snapped out of it," and after six years she now holds a responsible position, and many who meet her are unaware that she has any hearing defect. No. 2 shows slight improvement in the right and possibly a slight improvement in the left. All three are going about their work successfully. No ear treatment was given at any time.

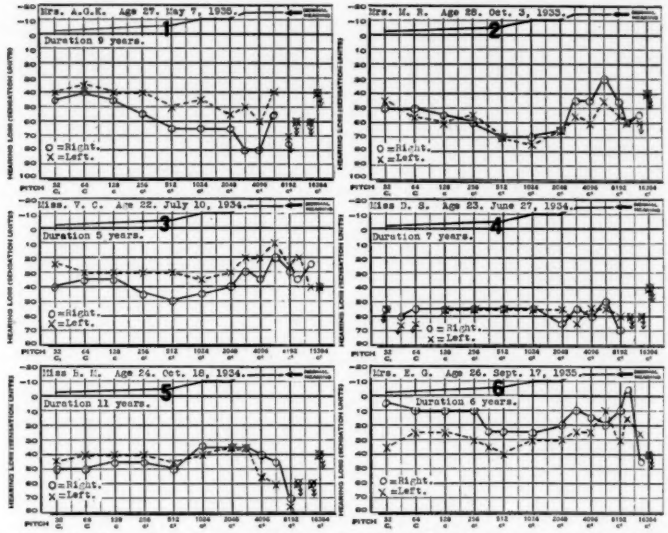

Fig. 10. Records of six patients in the third decade. None of these have as marked loss as No. 2 in Fig. 9.

Fig. 7 shows the records of three patients whose deafness has progressed subjectively and according to the audiometric tests. No. 1 shows as much as 35 dcb. additional loss in the left ear for tones of high pitch. The change in the left ear of No. 2 occurred in two months. (This patient's complete record, except for the test on July 21, 1932, is shown in Fig. 1.) Each successive test of the left ear of No. 3 showed slight increase in the hearing loss, and the two ears had reached almost the same level at the time of last test.

Fig. 8 shows the records of two patients whose hearing definitely improved during the time they were under observa-

tion. In No. 1 the improvement noted in the left ear between March 2, 1932, and Feb. 6, 1934, was quite marked, but by April 29, 1938, it had again fallen almost to the same level as it was in 1932. The lady whose record is given in No. 2 thought her hearing in the right ear was normal on Dec. 10, 1935, but subsequent tests showed that it was again falling. No. 1 was a diabetic and had bad teeth and tonsils. While she was hospitalized for the diabetic condition, she had an acute attack of cholecystitis, and at the time of operation a cyst,

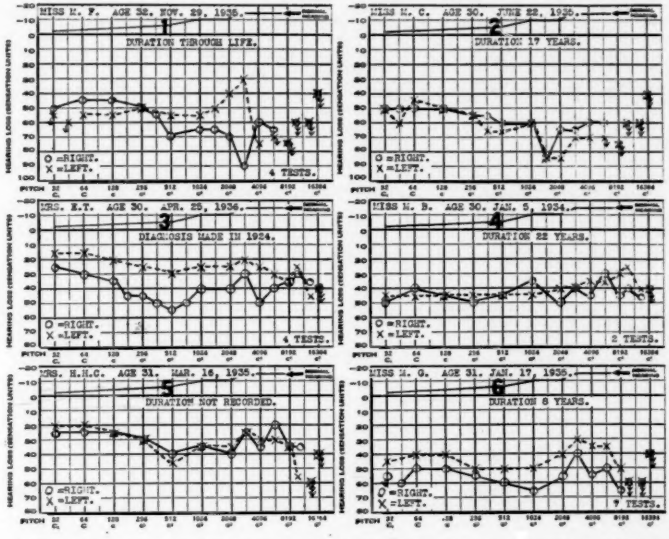

Fig. 11. Records of patients in the fourth decade. No. ' was first diagnosed in 1924.

the size of a lemon, in her liver was drained. No. 2 underwent a severe abdominal operation.

The patients whose records are shown in Fig. 9 were all under age 20 years. A great variation in acuity will be noted. No. 2 has a marked loss, especially for high tones, and has brittle bones. No. 4 also has brittle bones and has very little loss in the left ear. It might be said that the records of Nos. 3, 5 and 7 are similar, in that they show almost equal losses for all tones.

Fig. 10 shows the records of six patients in the third decade. No. 6 has a slight loss in the left ear, while No. 2 shows marked bilateral loss. The variation here is as great as that shown in the younger group.

Fig. 11 shows the records of three patients in their fourth decade. The losses in Nos. 3 and 5 are not great, while Nos. 1 and 2 are quite marked. The diagnosis of otosclerosis in No. 3 was made when she was tested with an early pitch range audiometer in 1924. No comparisons of the two records is

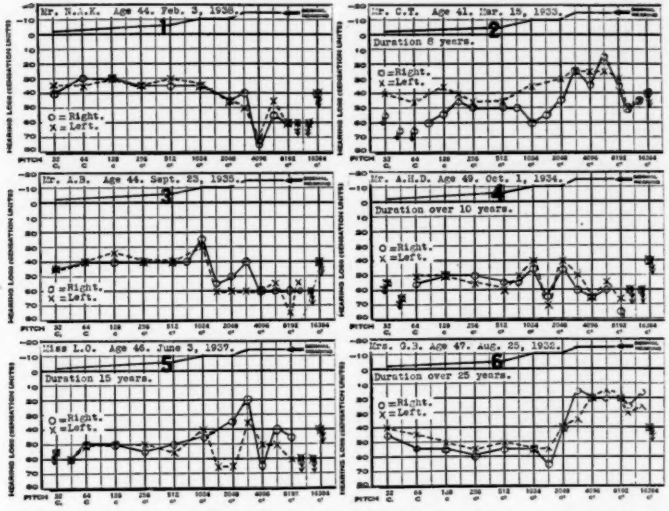

Fig. 12. Records of patients in the fifth decade. Some of these have no greater hearing loss than those shown in Fig. 9.

possible but the slight loss shown in her last test indicates that the hearing loss has been very little in the 12 years between the tests. In 1924, when she was unmarried, she was informed as to the generally accepted opinion concerning the influence of child-bearing upon the progress of deafness in otosclerotic mothers. In 1936, she had married but had remained childless and made inquiries as to the more recent thought concerning this subject.

Fig. 12 shows the records of six patients in their fifties. Losses equal to that shown in Nos. 1 and 2 can be matched in

the records of those in the younger groups. No. 2 shows less hearing loss than No. 6 of the 20-year group, yet she stated that she had noted her deafness for 50 years. No. 3, who is age 51 years and has been hard-of-hearing all his life, gives a record which shows no greater loss than No. 2, of Fig. 9, who is but age 18 years.

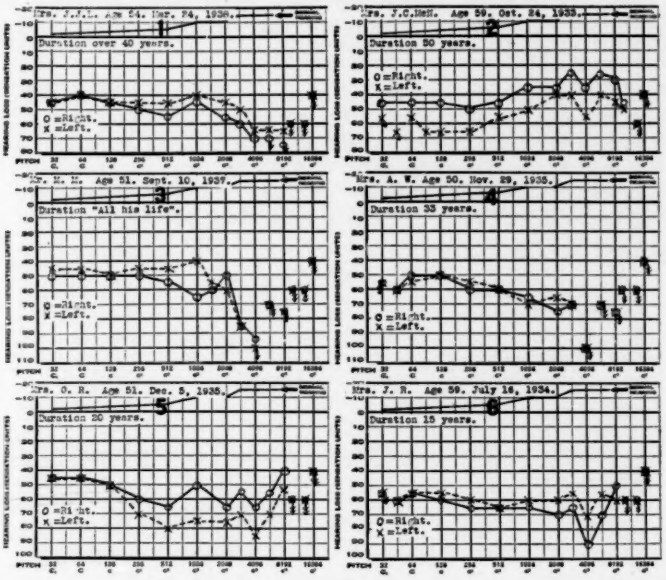

Fig. 13. Records of patients in the sixth decade. No. 2 shows no greater loss than some of those in the second decade but she has been aware of her hearing loss for 50 years.

The records of most marked deafness which have been discovered in this study are illustrated in Fig. 15. Nos. 4 and 6 are under age 25 years. No. 2 was age 71 years.

During the course of this study, the writer has been fortunate enough to be able to test at intervals the hearing of six women who have borne children during that time of observation. Three of these are shown in Fig. 14. All three were primiparas. The right and left ears are recorded separately for convenience of comparison. Three tests are recorded on each chart, one before childbirth; a second, shortly after, and the third at a later date. It is difficult to

see in any where a definite increase in deafness has accompanied the child-bearing.

Fig. 15 contains the records of three more women who have been tested before and after child-bearing. The manner in which the curves cross and recross shows that litle, if any, loss has occurred.

The six records shown here are insufficient evidence to prove or disprove the statement that pregnancy causes an

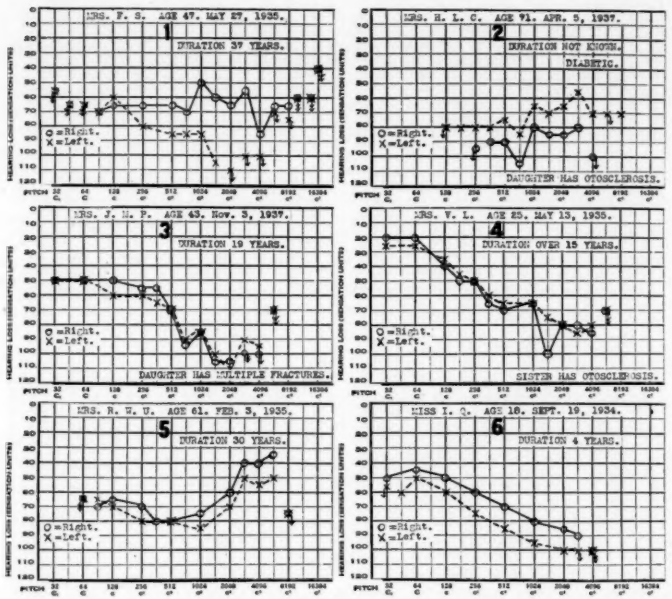

Fig. 14. Records showing the most marked hearing loss encountered during this study. No. 6 was age 18 years, while No. 2 was age 71 years.

increase in the deafness of otosclerotic members. Many more cases should be studied. In these six the deafness has not progressed. No other audiometric studies were available for comparison.

COMMENTS.

The short periods of time during which these patients have been under observation, *viz.*, up to six years, cover such a

small part of the lifetime of any one of them that no comprehensive conclusions may be drawn. It can be definitely stated that in some there was no measurable increase in the hearing loss. With those in whom the deafness has increased, no obvious factor in the physical condition or state of general health and no peculiar result in the functional tests gave clues to reveal whether or not a change in hearing might be predicted. This is evidenced in the fact that changes, either improvement or regression, were often unilateral. It so happens that

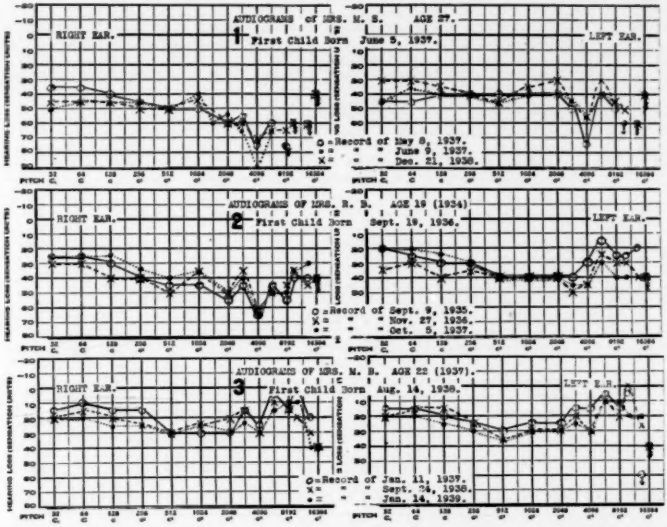

Fig. 15. Records of three mothers taken before and after their first babies were born.

the two patients which showed the most marked improvement underwent serious surgical operations, but since this study was conducted in a hospital it is to be expected that some of those under observation would be hospitalized.

It appears certain that time alone is not the factor determining the progress of the deafness.

The statements of patients relative to the progress of deafness cannot be accepted as factual unless the changes be quite marked. Often these patients claimed changes either of improvement or regression which the tests failed to substan-

tiate. The records of tests made when a patient felt "low" were often somewhat lower than those secured when he felt physically fit, or, as Holmgren stated, when he "willed to hear."

A rather peculiar vicious cycle of circumstances was frequently noted concerning the patient's perception for conversation, circumstances which may not be common to otosclerotics alone. When they are fatigued, they exert additional physical and nervous energy to understand their associates.

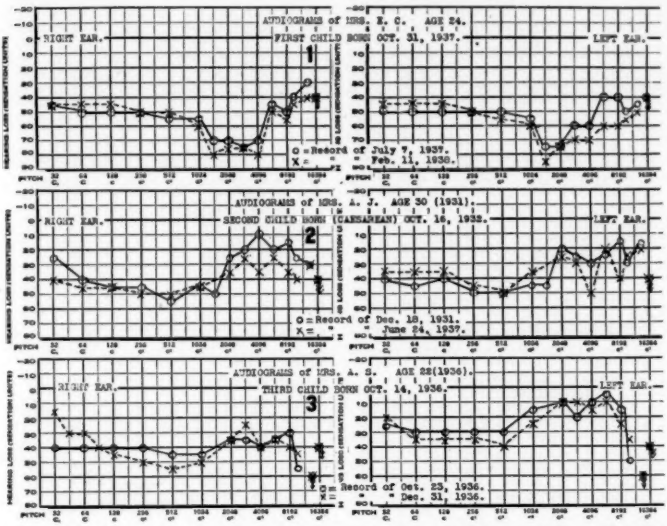

Fig. 16. Records of three mothers who bore children while this study was in progress. No. 3 was first tested after her third child was born.

This results in greater fatigue and eventually they cease to listen and, consequently, give the impression of greater loss than actually exists.

The evidence presented covers this subject very inadequately. No definite conclusions applicable in an individual case has been formulated. It is certain in some cases that no increase in deafness has occurred. In a few, the deafness has increased. To counteract this, the hearing has improved in some. In no case did the deafness progress to complete loss for the human voice. In his contact with otosclerotic patients,

the otologist should consider the possibilities very carefully before making his prognosis. Each individual case is worthy of the most careful study.

BIBLIOGRAPHY.

1. LEDERER, F. L.: Diseases of the Ear, Nose and Throat, p. 130, F. A. Davis Co., Philadelphia, 1938.

2. KERRISON, P. D.: Diseases of the Ear, p. 251 ff., J. B. Lippincott Co., Philadelphia, 4th Ed., 1930.

3. HOLMGREN, GUNNAR: Nelson's Loose Leaf Surgery of the Ear, p. 410. Edited by S. J. Kopetzky. Thos. Nelson and Sons, New York, 1938.

4. SUGGIT, STEPHEN: Observations on the Variations in Hearing in Otosclerosis. *Jour. Laryngol. and Otol.*, 53:5:294, May, 1938.

CASSELBERRY FUND.

A sufficient sum having accrued from the Casselbery Fund for encouraging advancement in the art and science of laryngology and rhinology, said sum is now available, in part or as a whole, for a prize award, decoration or the expense for original investigation or research in the domains mentioned above. Theses or reports of work must be in the hands of the Secretary before Feb. 1, 1940. Dr. Charles J. Imperatori, Secretary, American Laryngological Association, 108 East 38th street, New York.

THE FREQUENCY OF INCREASED NYSTAGMUS TIME AFTER ROTATION IN MULTIPLE SCLEROSIS.*†

DR. PAGE NORTHINGTON, New York.

Although our knowledge is considerable on the diseases that cause a hypoactive type of vestibulo-ocular reflex, information is meagre on the diseases that are responsible for the alteration in the reflex when it is hyperactive. For some time it had been my impression that a prolonged postrotatory nystagmus time occurred with greater frequency in those patients with the diagnosis of multiple sclerosis than in any other diseases, so it was thought to be worth while to obtain more information on its relative frequency by a survey of the records of patients who had functional ear examinations. Through the co-operation of the neurological service over several years, it has been possible to perform the cochlear and vestibular tests in a large group of patients, and this report represents a survey of a few over 2,000 case records.

REVIEW OF LITERATURE.

The accumulated literature of vestibular reactions in intracranial diseases is extensive but only a few reports were found on observations made in multiple sclerosis and other degenerative types of brain lesions. Barany reported a case of multiple sclerosis with findings that corresponded to those usually present in patients with acoustic neuroma. Brunetti reported a very similar case, in that the symptoms on the functional ear examination were suggestive of an VIIIth nerve tumor. Beck reported a case of unilateral temporary loss of cochlear and vestibular functions in a case of multiple sclerosis. In a patient with arteriosclerosis, Beck observed vertigo and nystagmus with falling backward during an attack. The hearing was normal. The nystagmus reaction was normal on the rotation and caloric tests, but the pointing and falling reactions were absent. He thought that this patient had small

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hemorrhages from arteriosclerosis in the cerebellar cortex. Leider thought that the spontaneous nystagmus present in a patient with syringomyelia was the result of a lesion of the arcuate fibres running from the lowest portion of the vestibular nerve. Poston cited the great frequency that nystagmus and other vestibular phenomena occurred in encephalitis and the considerable variations that might be in different patients. Friesner, in a group of 10 cases of multiple sclerosis, found that the spontaneous nystagmus frequently persisted despite the superimposed induced nystagmus, "so that in response to cold, caloric stimulation, one may have a normal nystagmus directed to the side opposite to the ear that is stimulated and yet the spontaneous nystagmus in some other direction will persist, and may even be accentuated." In one case of Friesner's there was no nystagmus, but normal vertigo and past-pointing on the caloric test. Spontaneous nystagmus occurred

TABLE I

POST-ROTATORY NYSTAGMUS TIME IN THE NORMAL

POST-ROTATORY NYSTAGMUS TIME IN SECTION FOLLOWING ROTATION OF TEN TIMES IN TWENTY SECONDS					
CASES	20 to 25	26 to 30	31 to 35	36 to 40	41 to 45
334	2 6 %	48 96 %	33 33 %	11 11 %	0 0 %

AVERAGE POST-ROTATORY NYSTAGMUS TIME WAS 28.4 SECONDS

in seven of the 10 cases with no regularity of direction, and in some there was a combination of directions. As determined by the pointing test, there was no vertigo in any of the cases. In none was there found any impairment of hearing. The rotation test was not done in Friesner's cases. Barre reported that the sudden onset and type of vestibular disorder in the early stage of some cases of multiple sclerosis might be thought to be an acute labyrinthine disease. He concluded from the rotation and caloric tests that the vestibular reactions were normal in 60 per cent, hyperactive in 11 per cent, and hypoactive in 9 per cent. The hyperactive group were in the well advanced cases of multiple sclerosis. Although he gave ony 11 per cent in the hyperactive group, he stated that the most outstanding finding of the vestibular symptoms in multiple sclerosis is the great predominance of subjective and irritative phenomena over deficiency.

METHOD OF EXAMINATION.

A physical examination of the ears, nose and throat was made. After making observations for a spontaneous nystagmus in both the face-front and face-up positions, the patient in the face-front position with eyes closed was rotated to the right, 10 times in 20 seconds. On stopping the chair — a brake-stop was not used — the patient was directed to open his eyes and look straight away in space. The postrotatory nystagmus was observed as to its direction, plane, amplitude and duration. Following rotation to the right, the patient was rotated to the left and similar observations were made. It was not particularly difficult to accurately determine the postrotatory nystagmus time in patients that had a spontaneous nystagmus. A spontaneous nystagmus was rarely found when the eyes were in the primary position, and when it did occur

TABLE II

POST-ROTATORY NYSTAGMUS TIME IN MULTIPLE SCLEROSIS, PSYCHONEUROSIS, ARTERIOSCLEROSIS AND INTRACRANIAL TUMORS

DIAGNOSIS		DURATION OF POST-ROTATORY NYSTAGMUS TIME IN SECONDS							
		-31	31to35	36to40	41to45	46to50	51to55	56to60	+60
MULTIPLE SCLEROSIS	52 CASES	32	5	6	22	3	3	5	5
PSYCHONEUROSIS	105 CASES	85	7	8	3	0	0	0	2
ARTERIOSCLEROSIS	60 CASES	47	5	4	2	0	1	0	1
INTRACRANIAL TUMORS	180 CASES	122	17	15	3	7	1	3	4

a point of fixation that moved the eyes from the primary position could be found that caused the spontaneous nystagmus to stop. Having determined the direction of gaze in which there was no nystagmus, the patient, following rotation, was told to fix again his gaze in that direction, for observation on the duration of the postrotatory nystagmus. The frequency of spontaneous nystagmus in patients with a postrotatory nystagmus time over 30 seconds is shown in Table III. An upper limit of 30 seconds for the duration of the normal postrotatory nystagmus time was used. An explanation for my adopting a lower limit than conventionally is used is given in the discussion.

CLINICAL MATERIAL.

With a few exceptions, the case reports are of patients that were examined in the hospital on neurological service, where they had been admitted for an investigation of their

illness. The diagnosis of the cases of intracranial tumors was made from the findings on operation or necropsy. The diagnosis in the cases, other than the tumor group, was made after examinations during a period of observation by the neurologists. The cochlear and vestibular tests were made as part of the general neurological investigation. It is seen in Table IV that the postrotatory nystagmus time was over 30 seconds in 77 per cent of the patients with multiple sclerosis, 19 per cent with psychoneurosis, 22 per cent with arteriosclerosis, and 27 per cent with intracranial tumors. In addition to those groups of diseases in which the postrotatory nystagmus time occurred with considerable frequency, it was found in at least one patient with the diagnosis of encephalopathy, encephalitis, encephalomalacia, posterior lateral sclerosis, amyotrophic sclerosis, syringomyelia, olivopon-

TABLE III

FREQUENCY OF SPONTANEOUS NYSTAGMUS IN PATIENTS WITH POST-ROTATORY NYSTAGMUS TIME OVER 30 SECONDS

DIAGNOSIS	POST-ROTATORY NYSTAGMUS TIME OVER 30 SECONDS	SPONTANEOUS NYSTAGMUS
MULTIPLE SCLEROSIS	40 CASES	+29 CASES -11 CASES 72 % 28 %
PSYCHONEUROSIS	20 CASES	+11 CASES - 9 CASES 55 % 45 %
ARTERIOSCLEROSIS	13 CASES	+ 5 CASES - 8 CASES 31 % 69 %
INTRATENTORIAL TUMORS	30 CASES	+ 6 CASES -24 CASES 20 % 80 %
SUPRATENTORIAL TUMORS	21 CASES	+14 CASES - 7 CASES 67 % 33 %

tocerebellar atrophy, general dysplasia of the nervous system, thrombosis of inferior cerebellar artery, cerebral thrombosis, neurofibromatosis, concussion of the brain, chronic lymphatic leukemia and syphilis. Those diseases may be classified in general as caused by degenerative types of lesions.

The frequency of spontaneous nystagmus as compared with that of prolonged postrotatory nystagmus in the case reports is shown in Table IV. The spontaneous nystagmus was present in 63 per cent of the patients with multiple sclerosis, 18 per cent with psychoneurosis, 18 per cent with arteriosclerosis, and 42 per cent with intracranial tumors. Although caloric tests were done, a statistical report of the results is not included here. It seems sufficient to state that in the 52 patients with multiple sclerosis, the postrotatory time was over 30 seconds in 40 cases. but the caloric nystagmus reaction occurred earlier than the normal time after beginning

the irrigation in only six cases. At least in this group of cases one must conclude that there is no parallel in the increase of nystagmus response to the rotation and caloric tests. In Tables V, VI, VII and VIII it is seen that the average hearing of the patients with multiple sclerosis, psychoneurosis, arteriosclerosis and intracranial tumors (cases of tumors of the cerebellopontine angle are not included) compares favorably in the different age groups with the normal hearing as reported by Bunch.

Since a visual disorder is reported commonly in multiple sclerosis and as it might be the cause of a complaint of dizziness or disturbance of gait, it would seem worth while to include its frequency in these case reports. The complaint

TABLE IV

SPONTANEOUS NYSTAGMUS AND POST-ROTATORY NYSTAGMUS TIME IN MULTIPLE SCLEROSIS, PSYCHONEUROSIS, ARTERIOSCLEROSIS AND INTRACRANIAL TUMORS

DIAGNOSIS	CASES	S. N.	DURATION OF POST-ROTATORY NYSTAGMUS TIME		
MULTIPLE SCLEROSIS	52	33 CASES 63 %	UNDER 31 SECONDS	12 CASES 23 %	OVER 31 SECONDS 40 CASES 77 %
PSYCHONEUROSIS	106	19 CASES 18 %	UNDER 31 SECONDS	86 CASES 81 %	OVER 31 SECONDS 20 CASES 19 %
ARTERIOSCLEROSIS	60	11 CASES 18 %	UNDER 31 SECONDS	47 CASES 78 %	OVER 31 SECONDS 13 CASES 22 %
INTRACRANIAL TUMORS	182	77 CASES 42 %	UNDER 31 SECONDS	132 CASES 72 %	OVER 31 SECONDS 50 CASES 27 %
LOCATION OF TUMORS:					
1.SUPRATENTORIAL	101	17 CASES 16 %	UNDER 31 SECONDS	71 CASES 70 %	OVER 31 SECONDS 30 CASES 30 %
2.SUBTENTORIAL:	81	60 CASES 74 %	UNDER 31 SECONDS	61 CASES 75 %	OVER 31 SECONDS 20 CASES 25 %
A.CEREBELLO-PONTINE ANGLE	40	30 CASES 75 %	UNDER 31 SECONDS	32 CASES 80 %	OVER 31 SECONDS 8 CASES 20 %
B.OTHER POSTERIOR FOSSA	41	30 CASES 73 %	UNDER 31 SECONDS	29 CASES 97 %	OVER 31 SECONDS 12 CASES 40 %

S. N. SPONTANEOUS NYSTAGMUS

with reference to the vision was that of either blurred vision, diminished vision or double vision, and it occurred in 14 of the 52 patients with multiple sclerosis. In only one had there been made the diagnosis of retrobulbar neuritis, and in this patient a temporary improvement in the vision followed an operation on the ethmoid and sphenoid sinuses.

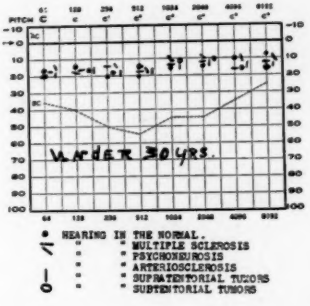
As to the complaints of the patients with multiple sclerosis that might be considered as a symptom of a vestibular disorder, 10 gave a history of having either dizziness, dizzy attacks or vertigo, some with nausea and vomiting, and 34 gave a history of a disturbance of equilibrium variously described as either unsteadiness, swaying, staggering, lack of balance, difficulty in walking, weakness in legs, stiffness in legs, drunken walk or wabbling gait.

Although a focus of infection is included as an etiologic factor in one of the many theories of the cause of multiple sclerosis, there was no evidence from the ear, nose and throat examination to support that theory in this group of patients.

DISCUSSION.

The important matter for consideration in this report is the diagnostic significance, if any, of the finding of postrotatory nystagmus time over the normal duration, so first, although it may be inexact, a standard time for the upper limit of the normal postrotatory nystagmus time must be adopted as a means of measurement. It is seen in this report that a nystagmus time of over 30 seconds is considered over the normal nystagmus time following rotation. There will be no general agree-

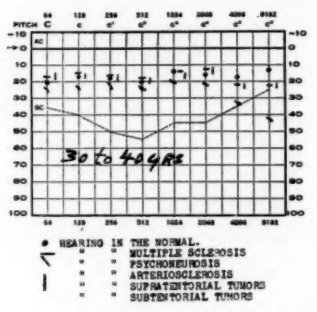
TABLE V



ment, I am sure, of my adopting such a short physiological duration for the postrotatory nystagmus time. The duration of the normal postrotatory nystagmus time is commonly given as 36 seconds for the upper limit, although the army adopted 34 seconds as their upper limit for acceptable aviation personnel. The complex nature of the vestibulo-ocular reflex makes it impossible to be able to obtain such precision measurements that a very definite limit can be established between the normal nystagmus reaction and the increased nystagmus reaction that may be applicable in every patient. It would be more accurate to designate reaction time as averages in the presumably healthy than to adopt absolute limits for the normal range. In addition to the variables inherent in the rota-

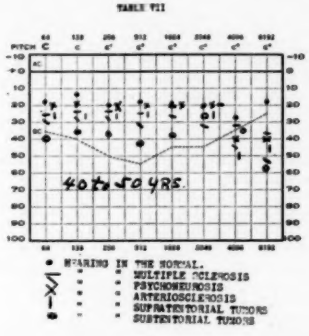
tion test, lack of precisely the same head position and variations in acceleration, retardation, timing and observation for nystagmus from patient to patient, there are doubtlessly incalculable influences on the nystagmus by the central nervous system even in the normal. To see one case of voluntary nystagmus: a person who at will can begin and stop his eye movements, which in all respects appeared to be similar to a nystagmus that might be caused by disease, the examiner is not likely to consider the vestibulo-ocular reflex as being entirely involuntary in character. That an emotional status might vary the postrotatory nystagmus time is suggested by Mowrer in his studies of nystagmus in pigeons during excitement and during rest. On observations for spontaneous nystagmus fol-

TABLE VI



lowing brain lesions, made by Barrera in monkeys, the nystagmus for a few days after subsiding could be made to reappear on excitement; possibly it was fear or anger. In practice, the decision of whether a reflex action is either slightly hypoactive or slightly hyperactive or just within the normal limit largely depends on the examiner's clinical experience. Several years past, I examined some 500 midshipmen at the Naval Academy as a survey for potential aviation personnel. The rotation test was a part of the examination, and the nystagmus time following rotation in 104 of those midshipmen was obtained from the files of the Bureau of Medicine and Surgery, through the courtesy of Comm. J. C. Adams. It may be seen in Table I that the postrotatory nystagmus time was not over 30 seconds in any one of the 104 midship-

men that were examined. Consistent with the low upper limits for postrotatory nystagmus time in Table I is the report by Patson and Segar, in which the nystagmus time was over 30 seconds in only three cases out of the 100 student aviators examined. Malan reports that in the examination of 11,000 normal subjects the postrotatory nystagmus was over 30 seconds in only 7.12 per cent. Guilder and Hopkins found a mean of 19.6 time for postrotatory nystagmus time in a normal group, which is almost identical with the findings listed in Table I, and that the maximum time found in any case was 34 seconds. The mean duration of postrotatory nystagmus time is given by Barany as 25 seconds, and by Jones as 24 seconds. In the examination of those in health and illness, a

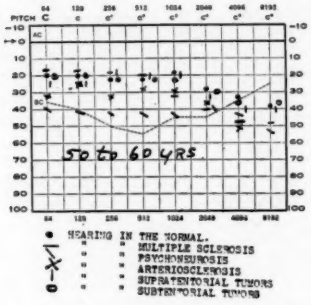


postrotatory nystagmus time of over 30 seconds has been found in only a few persons when there were no substantiating symptoms of some disease; therefore, 30 seconds was used as the upper limit for normal postrotatory nystagmus time.

It would be of considerable clinical aid in the evaluation of the symptom of prolonged postrotatory nystagmus time in patients if our knowledge of vestibular physiology was more complete. There is no general agreement among research and clinical investigators, even on a theory to account for nystagmus continuing after the rotation of the subject ceases. The unsettled problem is that of whether the nystagmus after rotation is dependent on the stimulation continuing from temporary alterations in the vestibular receptor caused by retard-

ation and stopping of the rotation, or the nystagmus is dependent on some after-discharge process of impulses built up in the vestibular nuclear complex and connections during this period of stimulation. Reports of observations are plentiful in support of a theory for either a peripheral or central vestibular responsible mechanism. It may well be, as Mowrer concludes from a discussion of the evidence presented by research studies in the field of physiological vestibular function, that both the vestibular receptor and vestibular nuclei are capable, by quite independent mechanisms, of after-discharge. If the hypothesis is accepted that either one of the two special mechanisms may be responsible for the physiological nystagmus reaction after rotation, there arises the neces-

TABLE VIII



sity in clinical medicine of determining whether the cause of variations from normal nystagmus reactions is from disease of the peripheral or central vestibular components in each patient examined. In the clinical material that is reported on here, the prolonged postrotatory nystagmus time occurred chiefly in two groups of patients; one with lesions of a degenerative type, and one with neoplastic lesions. It must be considered as possible for intracranial tumors to modify the vestibulo-ocular reflex, either by alterations in the brain at the site of the tumor or by pressure effects on the intracranial structures or on the inner ear. Although some claims have been made by observers for a hypovestibular reactivity as a result of general increased pressure, there is no mention made of a hypervestibular reactivity as shown here by the pro-

longed postrotatory nystagmus time in 27 per cent of the intracranial tumors. Hughson reported that when the spinal fluid pressure was raised in cats by intravenous injections of hypotonic solutions there was also an elevation of the intralabyrinthine pressure. If it be conceded that there is increased intralabyrinthine pressure in patients with intracranial tumors from Hughson's observations, with the obvious objections to such a conclusion, still there is no evidence that such an alteration in the vestibular end-organ would be responsible for a prolonged postrotatory nystagmus time. In the examination of patients with hydrocephalus, in which the interpretation of functional ear examination was not complicated by having to consider the local effects of the brain tumor, an increase in the vestibulo-ocular reflex was never seen. Therefore, it does not seem likely that the prolonged postrotatory nystagmus time in the intracranial tumor cases is due to any changes in the vestibular end-organ that might be the result of increased intracranial pressure. When an intracranial tumor involves the acoustic nerve, a gross loss of both vestibular and cochlear reactivity on the side of the lesion to caloric testing is regularly found, but the rotation test may not show a corresponding hyporeactivity of the vestibulo-ocular reflex. In two of the cases of acoustic neuroma in this report there was actually a prolonged postrotatory nystagmus time, the cause of which is certainly not likely the peripheral vestibular disorder. In tumors of the posterior fossa other than those of the cerebellopontine angle, a prolonged postrotatory nystagmus time was found in 44 per cent. My opinion is in agreement with that reported by Fisher on the effect of increased intracranial pressure on the vestibular reactions, which is that abnormal vestibular findings do not indicate generalized increased intracranial pressure, but since they are not always produced by actual destruction, it must be some pressure effect of a local nature.

Even in the patients with degenerative diseases of the central nervous system, there is no proof that some alteration in the peripheral vestibular component is not a causative factor in abnormal vestibular reactions. Beck reported three cases of multiple sclerosis with variations in the cochlear and vestibular reactivity on the several examinations made. In one of his patients, he found total loss of cochlear and vestibular

reactivity to tests in the left ear, and on examination a week later the hearing was five metres for spoken voice, and vestibular reactions were hyperactive on the caloric and rotation tests. Beck considers this variation in the cochlear and vestibular functions as analogous to temporary blindness at times found in patients with multiple sclerosis. Barany reported a case of multiple sclerosis with autopsy finding that was of particular interest because the loss of cochlear and vestibular functions on one side had led to the diagnosis of an VIIIth nerve tumor and an operation. The finding in these cases of cochlear and vestibular impairment warrants the opinion that there was a peripheral lesion. The interesting question is: Was the cause peripheral or central in Beck's case, to account for the hyperactive vestibular reactions found on the second examination? Since Beck and Barany made these few cases of multiple sclerosis the subject for special reports, the symptoms the patients had were most probably exceptional. At least, in the 52 cases of multiple sclerosis in this report, there was no history in any patient of temporary deafness or was there a considerable loss of cochlear or vestibular function found on examination. Friesner reported normal hearing in 10 patients with multiple sclerosis. Although one may not be able to exclude a lesion of the peripheral vestibular component in multiple sclerosis or brain tumors in these patients, the following reasons seem to justify attributing the cause of the prolonged postrotatory nystagmus time to changes in the central nervous system.

1. The consistently good cochlear function that was present. It is seen in Tables V, VI, VII and VIII that the hearing compared favorably with that of the normal group. It was my impression on examining patients with multiple sclerosis to expect excellent hearing as one of the findings. Some impairment of cochlear function is found regularly in peripheral vestibular disorders.

2. The hyperactive type of nystagmus response that was found on the rotation test. A hypoactive nystagmus response has been the only type observed by me in lesions that were known to be limited to the peripheral vestibular component, either in the end-organ or VIIIth nerve.

3. The spontaneous nystagmus present was of central type. That considerable evidence along with that of the primary

site of the pathology in multiple sclerosis and brain tumors would seem to warrant the opinion that the cause of the prolonged postrotatory nystagmus is in the central nervous system. As to whether this increased vestibular reactivity is caused by an irritative lesion in the central vestibular components or is due to a lesion inhibiting normal control of centres over the vestibular ocular reflex, I am inclined to the latter opinion, which also is that of Unterberger. In support of this idea, a prolonged postrotatory nystagmus time has been observed rather consistently by me in experimental animals, cats and monkeys, following extensive operative lesions in the cerebellum and sometimes after cerebral lesions when the vestibulo-ocular tracts were not involved. To the clinician, the important answer is wanted to the question of the significance, if any, of the increase in the vestibulo-ocular reflex on the rotation test. If that symptom be taken alone from the examination of a patient it would seem from the findings reported in Table IV that it most probably signifies a disorder of the central nervous system and, in the order of its frequency, either multiple sclerosis, brain tumor, arteriosclerosis, psychoneurosis or possibly one of a considerable group of diseases of a degenerative type. Any estimation except by a neurologist that might be made from a survey of the case records of the relative importance of this symptom in the diagnosis that was arrived at is of questionable value; however, it did not appear that the finding of a vestibular disturbance was of any great helpfulness in arriving at a diagnosis of the intracranial diseases. Particularly was this true in patients with brain tumors. Although it has been very rare for a patient with an intracranial disease to show signs of only a vestibular disorder on the neurological investigation, it occasionally did happen. As an example, I want to cite the case of a patient not included in this report, who had a total loss of vestibular reactivity on one side, along with good hearing, as the only finding, indicating an organic brain disease. He was discharged from the hospital with the diagnosis of psychoneurosis, to return at the end of seven weeks with well developed general neurological symptoms of a cerebellar tumor. Although the postrotatory nystagmus time was prolonged in 44 per cent of the cases of posterior-fossa tumors, excepting those of the cerebellopontine angle, it is doubtful that this finding was of any real value in the diagnosis because

of other dependable neurological symptoms that were found. The patients with arteriosclerosis did not appear as a rule to present a difficult diagnostic problem. It should be cited, however, that usually patients with arteriosclerosis showed a considerable fluctuation in the reactions on vestibular tests when examined on different occasions. My chief purpose in reporting these observations was to present the finding of a prolonged postrotatory nystagmus time in 77 per cent of the cases of multiple sclerosis, and in 19 per cent of the cases of psychoneurosis. The great frequency of this finding in multiple sclerosis is conspicuous as a nystagmus symptom as it may be seen that spontaneous nystagmus, which is recognized as a diagnostic symptom of multiple sclerosis, occurred in only 63 per cent of the cases. It is thought that any symptom occurring with the frequency of 77 per cent in multiple sclerosis might be of importance in the diagnosis because this disease, as Wechsler states, is not characterized by a definite clinical syndrome but is one in which the diagnosis depends upon the frequency and consistency of a considerable variety of symptoms. And the occurrence of prolonged postrotatory nystagmus time in 19 per cent of the cases with the diagnosis of psychoneurosis, some having spontaneous nystagmus, raises the question of the possibility of these cases being early multiple sclerosis or possibly some other organic disease. I am told by the neurologists that about 20 per cent of the patients with the diagnosis of psychoneurosis later develop symptoms characteristic of an organic disease of the central nervous system, frequently multiple sclerosis. It, therefore, would not be at variance with such clinical observations if the 19 per cent of cases with the diagnosis of psychoneurosis that had prolonged postrotatory nystagmus time did develop signs that lead to a change in the diagnosis to some organic disease. Before any opinion could be had on the increase in the vestibulo-ocular reflex action on the rotation test as a symptom of multiple sclerosis occurring earlier than those on which the diagnosis is commonly made, examinations would have to be made from time to time on that group of patients with the diagnosis of psychoneurosis. Dr. Dillenberg, of the Neurological Service, has attempted to follow up those patients with the diagnosis of psychoneurosis that had a prolonged postrotatory nystagmus time but so far with little reward for his efforts. Although he has been able to follow up for examina-

tion only two of the patients with the diagnosis of psychoneurosis that had a prolonged postrotatory nystagmus time, he is continuing this study.

SUMMARY.

1. Thirty seconds was used as the upper limit for normal postrotatory nystagmus time.

2. Prolonged postrotatory nystagmus time occurred in 77 per cent of the cases of multiple sclerosis, 19 per cent of psychoneurosis, 22 per cent of arteriosclerosis, and 27 per cent of intracranial tumors.

3. Spontaneous nystagmus occurred in 63 per cent of the cases of multiple sclerosis, 18 per cent of psychoneurosis, 18 per cent of arteriosclerosis, and 42 per cent of intracranial tumors.

4. The hearing of the patients with multiple sclerosis compared favorably with the hearing of a normal group.

5. An attempt to follow up for examination the group of patients with the diagnosis of psychoneurosis who had a prolonged postrotatory nystagmus time has not been successful.

CONCLUSIONS.

Prolonged postrotatory nystagmus time was a common symptom in 52 patients with multiple sclerosis, having occurred more frequently than spontaneous nystagmus.

REFERENCES.

BARANY, R.: Zur Differentialdiagnose zwischen Acusticustumor und multipler Sklerose mit bulbärem Sitz. *Monatsschr. f. Ohrenheilk.*, 47:693-694, 1913.

BARRE, J. A.: Topographic Syndromes of the Central Vestibular Pathways in Man. *Rev. d'oto-neuro-ophthalmol.*, 15:353, 1937.

BECK, O.: Anomalien der Zeigereraktion bei Arteriosklerose. *Internat. Centralbl. f. Ohrenheilk.*, 17:228, 1920.

BECK, O.: Gehörorgan und Multipler Sklerose. *Monatsschr. f. Ohrenheilk.*, 47:875-877, 1913.

BRUNETTI: Nystagmus Reaction in a Case of Multiple Sclerosis. *Rev. d'oto-neuro-ophthalmol.*, 5:935-943, 1927.

BUNCH, C. C.: Age Variations in Audition Acuity. *Arch. Otolaryngol.*, 9:625, 1929.

CAMIS, M.: The Physiology of the Vestibular Apparatus. Oxford Univ. Press, 1930.

FRIESNER, I.: The Symptoms of the Auditory and Vestibular Apparatus in Multiple Sclerosis. Multiple Sclerosis Sec., 3:95, Paul B. Hoeber, 1922.

GRIFFITH, C. R.: Concerning the Effects of Repeated Rotation Upon Nystagmus. THE LARYNGOSCOPE, Vol. XXX, Jan., 1920.

GUILDER, R. P., and HOPKINS, L. A.: Audition Function Studies in an Unselected Group of Pupils at the Clarke School for the Deaf. III. Relation Between Hearing Acuity and Vestibular Function. THE LARYNGOSCOPE, 46:3, 1936.

HOLSOPPLE, J. G.: Factors Affecting the Duration of Postrotation Nystagmus. *Jour. Compar. Psych.*, 3:283-304, 1923.

HUGHSON, W.: A Note on the Relationship of Cerebrospinal and Intracranial Pressure. *Amer. Jour. Physiol.*, 101:396, 1932.

JONES, I.: Equilibrium and Vertigo. Philadelphia, 1918.

LEIDLER, R.: Ueber die Beziehungen der Syringomyelis, resp. Syringobulbie, zum zentralen Vestibularapparat. *Ztschr. f. Ohrenheilk.*, 76:201-214, 1918.

MALAN, R.: Risultati della prova rotatoria in 11,000 sogetti normali. *Ann. li. Laryngol., Otol., Rhinol.*, Fasc. 2, 1926.

MOWRER, O. H.: Influence of "Excitement" on the Duration of Postrotational Nystagmus. *Arch. Otolaryngol.*, 19:46-54, 1934.

PARSONS, R. P., and SEGAR, L. H.: Barany Chair Test and Flying Ability. *Jour. A. M. A.*, 70:1064, 1918.

POSTON, R. I.: Vestibular or Labyrinthine Epidemic Encephalitis. *Brain*, 49:482-524, 1926.

UNTERBERGER: Vestibularis und Grosshirn zur Frage der Vestibularere, "Ubererregbarkeit." *Ztschr. Hals. u. Heilk.*, 38:221, 1935.

WECHSLER: Clinical Neurology. W. B. Saunders Co., 1927.

20 East 53rd Street.

A SURVEY OF THE USE OF SULFANILAMIDE IN ACUTE OTITIS MEDIA.*

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No one familiar with the literature of otitic meningitis since 1936, in which year three French physicians[1] reported the first recovery of streptococcic meningitis when prontosil was used, can fail to be converted to the use of sulfanilamide in such serious conditions. Striking, indeed, is the fact that reviews of the literature by Gray,[2] in 1935, and Anderson,[3] in 1937, produce records of no more than 76 recoveries of streptococcic meningitis in the previous 35 years. Contrasted with this is the ever-growing number of reported recoveries, now well over 200, all having occurred since Feb., 1936, in the relatively short period of three and one-fourth years. One is forced to give sulfanilamide and allied compounds the credit for this marked improvement in the mortality rate of this previously almost surely fatal condition.

These clinical facts, along with many reports of well controlled animal experiments, give incontrovertible evidence that in sulfanilamide is a weapon that, although not infallible, and not without dangers, deserves consideration in combating infections caused by the hemolytic streptococcus.

If results of this kind can be secured in the grave condition of meningitis, it is natural that the mind of the physician should turn to the disease that gave rise to the infection of the meninges and attempt to cure that disease before such infection takes place. Otitis meningitis of necessity calls for a previous aural infection, with spread from this focus directly or indirectly to the meninges. Two important questions then arise: *1.* Can acute otitis media be cured, controlled or favorably influenced by the use of sulfanilamide? *2.* Can the drug be used with sufficient safety to justify its use? The ideal method of answering these questions would call for the comparison of statistics from a large series of treated and control cases. A large orphanage or similar institution would

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lend itself better to this type of study than a children's hospital. In the former, treatment could be used at the onset of otitic symptoms, whereas in the latter, cases are admitted in all stages of the disease. In the absence of such data, and lacking the opportunity to study this disease in the method outlined above, I have attempted to secure some information by means of a questionnaire. This is by no means an ideal method and I am fully conscious of its weakness and limited value.

1. Do you use sulfanilamide in treating acute otitis media? Yes..... No..... All cases..... Early..... Late.....
2. Is this treatment used before the result of cultures is secured? .....
3. How long have you used sulfanilamide in the treatment of acute otitis media? .....
4. How many cases have been so treated?.....
5. Is this figure approximate?.....
6. Have you used control cases?.....
7. Do control cases have more complications than treated cases? .....
8. Do you think the use of sulfanilamide has reduced the number of cases of surgical mastoiditis?.....
9. Can you give accurate figures on the above question? .....
10. Do you use sulfanilamide in acute nasopharyngitis and coryza before the onset of ear symptoms?.....
11. Has this reduced the incidence of acute otitis media?.....
12. Have you had any serious complications resulting from sulfanilamide administration? .....
13. Do you take special precaution to avoid such complications? .....
14. What dosage of sulfanilamide do you use?
    For adult? .....
    For child? .....
15. Have you used M. & B. 693 (Sulpho-Pyridine) in otitis media? .....
16. Is it superior to sulfanilamide? If so, when? .....
17. General remarks .....
    .....
    .....

Signed.....

This questionnaire was submitted to each member of the American Otological Society, to five additional otolaryngologists in each State in the Union, and to 15 otologists in Canada. In addition, the questions were sent to about 150 outstanding members of the American Academy of Pediatrics. The questions asked were of necessity generalizations, because one cannot expect to get replies from a busy physician if the reply calls for looking up case records and submitting accurate statistics.

TABLE I.

Otologists		Pediatricians	
37 States	100	26 States	76
Canada	6	Canada	2
Total	106	Total	78
Total physicians	184		

Although about 500 questionnaires were sent out, only 184 containing usable material were returned. Fifty per cent of the pediatricians made replies, whereas only 25 per cent of the otolaryngologists were heard from. Geographically, the pediatricians represented 26 States and Canada, the otolaryngologists, 37 States and Canada (see Table I).

TABLE II.

	Pediatricians	Otologists	Total	%
Physicians who use sulfanilamide in acute otitis media	72	93	165	89.6
Physicians who do not use sulfanilamide in otitis media	6	13	19	10.4
Using the drug early	49	63	112	61.0
Using the drug in all cases	12	24	36	21.0
Using the drug late	7	11	18	18.0

Further analysis of the replies shows that of the 184 physicians who submitted data, 165, or 89.6 per cent, use sulfanilamide in the treatment of acute otitis media. Of these, 60 per cent believe that early use of the drug secured the best results. In addition, 58.5 per cent believe that early administration is of sufficient importance to call for administration prior to the identification of the infecting organism. In other words, before spontaneous perforation of the tympanic membrane

has occurred or myringotomy has been performed (see Tables II and III).

TABLE III.

	Pediatricians	Otologists	Total	%
Using the drug before cultures are secured	47	61	108	58.5
Using the drug only after cultures are secured	19	24	43	41.5

The number of cases treated by this group of physicians, both pediatricians and otolaryngologists, totals 9,667. The mortality rate for the series was 0.041 per cent, or one fatality for each 2,417 cases treated (see Table IV).

TABLE IV.

	Pediatricians	Otologists	Total
Number of cases treated	3,845	5,822	9,667
Number using control cases	22	24	46
Complications reduced in treated series	18	17	35
Complications not reduced in treated series	4	7	11

It was the opinion of 76 per cent of the physicians using the drug that the incidence of mastoiditis was definitely reduced by its administration; 8 per cent believed that the drug had no influence upon the development of the above complication, while 16 per cent were undecided (see Tables IV and V).

TABLE V.

	Pediatricians	Otologists
Believe sulfanilamide reduces the incidence of mastoiditis	73.0%	79.5%
Believe sulfanilamide does not reduce the incidence of mastoiditis	7.6%	8.5%
Doubtful as to value of sulfanilamide	19.2%	11.8%

The statistics secured regarding control series were meagre and inadequate. Only 46 physicians had any experience with control cases, and no series was large enough to permit the drawing of reliable conclusions. Of these men, 76 per cent believed that control cases developed more complications than

the treated cases, while 24 per cent did not believe complications were reduced.

Only 27 per cent of the 184 physicians used this drug in the treatment of acute nasopharyngitis before the onset of ear symptoms. Of these, half were of the opinion that the incidence of otitis was reduced, 40 per cent were doubtful, and 10 per cent felt that no favorable influence could be noted.

TABLE VI.

	Pediatricians	Otologists	Total
Serious complications reported	9	15	24
Fatalities	1	3	4
Complications reported			
Nonfatal			Cases
Severe glossitis			1
Acidosis and recurrence of infection			1
Encephalitic symptoms, temporary blindness			1
Acute hemolytic anemia			4
Nature of complications not stated			6
Severe jaundice			2
Leukopenia severe			1
Nephritis			1
Hepatitis			1
Hemolytic jaundice			1
Acute anemia			1
Total			20
Fatal			
Hemolytic anemia			2
Severe jaundice			1
Agranulocytosis			1
Total			4

Mortality rate 1 in 2,417 cases, or 0.041 per cent.

The fatal complications reported were but four in number. It was not definitely stated whether these were cases of uncomplicated otitis media receiving the relatively small doses usually advised for this condition. One of these fatalities was accompanied by severe jaundice, the other three being blood diseases, *ie.*, hemolytic anemia, two cases, and one of granulocytopenia. Twenty other nonfatal complications were reported (see Table VI).

Corr and Root,[5] in a recent article, point out that to date the smallest dose of sulfanilamide given preceding the onset of granulocytopenia was 25 gm. (375 gr.), and even in this case there was a probability that the patient had taken more. In all other reported fatalities, 35 gm. (475 gr.) or more had

been given over at least a 14-day period. From this they conclude that a total distributed dose of 20 gm. (300 gr.) is probably safe.

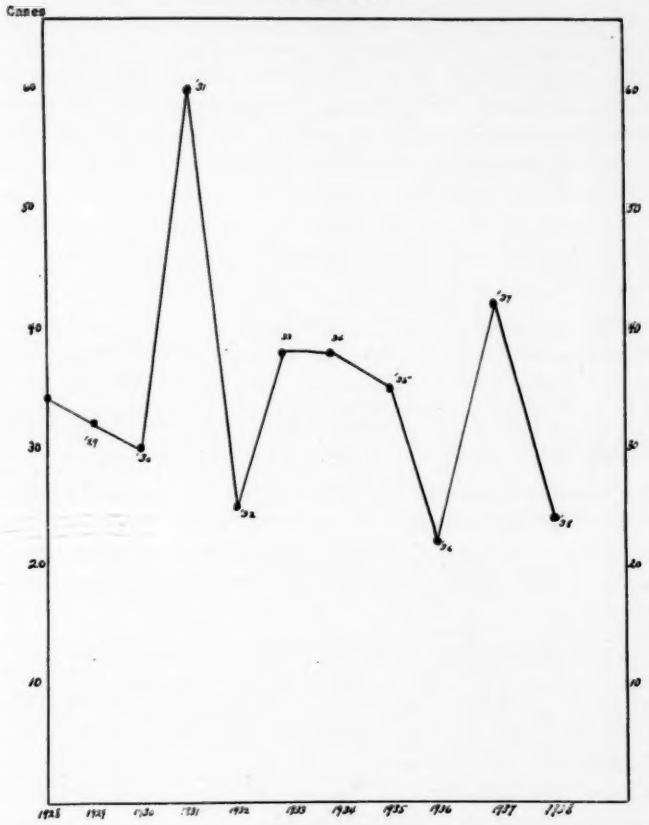
Although there was great variation in the dosage advised, the average administration was 0.75 to 1 gr. per pound of body weight for the first 24 hours. Afterward the dose was decreased if conditions permitted. There is a growing belief that blood concentration estimations are not as important as once supposed. In many individuals it is difficult to secure a 10 mg. per 100 cc. concentration without huge dosage, and good results are frequently secured in cases where the concentration was much lower. All, however, agree that frequent blood counts should be made to detect changes of the white or red blood systems.

An estimation of the incidence of acute surgical mastoiditis in our large city hospitals probably throws little if any light upon the value of sulfanilamide therapy. We know that if necrosis of the mastoid bone has occurred, sulfanilamide in any quantity will not cure this abscess. According to Lockwood,[4] the efficiency of sulfanilamide depends upon the ability of the blood stream to distribute blood to the infected parts. When central necrosis exists, circulation fails and only the peripheral portion of the lesion is reached by the drug. It is this fact which probably accounts for some of the atypical mastoid pictures that have been appearing since the beginning of the use of sulfanilamide. Most otologists have seen cases that were apparently cured except for persistent aural discharge, which upon operation proved to have well developed latent abscesses of considerable extent. The fact must be recognized that sulfanilamide therapy will not cure the mastoid infection that has undergone necrotic changes. Such conditions still demand surgery.

In the University of Pennsylvania Hospital in Philadelphia, the incidence of acute surgical mastoiditis has not changed appreciably in the last three years. The number of cases coming to operation during the past 11 years ranges from a high of 60, in 1931, to a low of 22, in 1936. In 1937 and 1938, there were 42 and 24 cases, respectively (see Table VII). Only six cases have been operated on prior to May 8, 1939. From these figures one concludes that preoperative adminis-

tration has not appreciably changed the curve of the incidence of acute surgical mastoiditis in this hospital. This, however, cannot be considered as a brief against sulfanilamide in the treatment of acute otitis. In our clinic, the cases are usually admitted with the disease well advanced and with

TABLE VII.



mastoid infection of considerable degree already established. Many have received no treatment whatever before coming to the hospital. Experience at the University Clinic in the treatment of these cases seems to support the idea of Lockwood that, although improvement may take place in the areas where adequate circulation is maintained, pre-existing necrosis cannot be improved.

The results of this survey do not permit the drawing of any sweeping conclusions regarding the value of sulfanilamide therapy in otitis media. Conclusive information still will depend on the results obtained from the comparison of a large series of control and treated cases; however, the following figures are interesting:

1. Almost 90 per cent of the physicians co-operating in the survey are using sulfanilamide in the treatment of acute otitis media. It is possible that most of the enthusiasts replied to the questionnaire, while those not using the drug or those having poor results failed to respond. In this event, the percentage of favorable replies would drop greatly had a full response been obtained.

2. Sixty per cent of the pediatricians and otologists who have had experience with sulfanilamide believe that its use reduces the incidence of acute surgical mastoiditis.

3. The majority believe that if used it should be used early, before mastoiditis has had a chance to develop.; 58.5 per cent use the drug before the results of cultures are secured.

4. In a series of 9,667 cases, a mortality rate of 0.041 per cent, or one death in 2,417, cases was reported.

5. In cases where the pneumococcus is either found or suspected, sulfapyradine is probably the drug of choice.

6. There is growing clinical evidence to support the idea that the blood concentration is not as important as previously supposed.

7. This survey shows that the use of sulfanilamide in acute otitis media is widespread, that its use is relatively safe, and that those using the drug are as a rule enthusiastic over the results obtained; this enthusiasm, however, lacks the supporting proof of control cases.

BIBLIOGRAPHY.

1. Causse, Loiseau, and Gisselbrecht: *Ann. d'oto. laryngol.*, 194, Feb., 1936.

2. Gray, H. J.: *Jour. A. M. A.*, 105:92, July, 1935.

3. Anderson, D. A.: *Jour. A. M. A.*, 108:1591, May, 1937.

4. Lockwood, John: Personal Communication.

5. Corr, P., and Root, R. N.: *Jour. A. M. A.*, 112:1939, May, 1939.

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SKULL FRACTURES INVOLVING THE EAR. A CLINICAL STUDY OF 211 CASES.*

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(Continued from August issue.)

OTOSCOPIC EXAMINATION.

The otoscopic examination should be made as soon after the injury as possible, particularly to detect hematotympanum or liquor tympanum, the evidence of which may be gone if the examination is deferred for several weeks. The signs seen by the otologist in his early examination may become very important in making the diagnosis and in evaluating the future course of the condition. Examination will reveal any signs of fracture of the walls of the external canal, some of which will surely disappear after the lapse of time. The evidences of a fracture of the external auditory canal are blood clots and bloody crusts on its walls, ecchymoses, subcutaneous hematomata, ridges or hollowing of the wall. These signs are more commonly encountered on the posterior or superior canal walls. If bloody crusts cover the skin surface, one may have to wait several days before the underlying condition can be visualized. An actual break of the skin is quite rare. In my series of 150 consecutive cases of head injuries seen at the Emergency Unit of the Milwaukee County Hospital, fractures of the external canal were encountered six times. Fracture of the external canal walls is never caused by transverse petrous fractures, nor any variety thereof. Longitudinal fractures are usually accompanied by a rupture of the membrana tympani and the escape of blood or spinal fluid from the ear. At this early examination the presence or absence of the spontaneous signs of vestibular disturbance, particularly spontaneous nystagmus, can be ascertained. Mygind,[1] Davis,[45] the author,[2] and others have stressed this early examination of head injury cases.

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HEMORRHAGE FROM THE EAR.

Hemorrhage from the ear after a head trauma is almost indubitable evidence of a basal skull fracture and of the longitudinal variety. Either one ear or both may bleed. Gurdjian[46] found this complication in 476 instances in a series of 2,600 cases of skull fracture seen at the Detroit Receiving Hospital in a five-year period but did not believe that this symptom always implied a fracture through the petrous bone. Besley[10] reports bleeding from the ear in 31.5 per cent of his cases, Voss[3] in 32 per cent, Wortis and Kennedy[47] in 34.2 per cent, Borden[48] in 75 per cent, Moody[49] in 24 per cent, and Ransahoff[50] in 31 per cent.

Gurdjian[46] and Yerger[51] believe that a rupture of the drum after head trauma is not necessarily an evidence of a basal skull fracture, but the majority of authors (Ramadier and Caussé,[13] Davis,[45] Besley,[10] Voss,[3] Phelps,[52] W. Lange[21] and Mellenger[4]) believe that bleeding from the ear after an injury to the head is a sure sign of temporal bone fracture. In the course of his operative work on these cases, Voss,[3] in agreement with other authors (Barnick, Linck, Lange, Ulrich, Brunner and Marx), could only find a torn drum when the margotympanicus had been reached by the fissure. When bleeding from an ear occurs, it is usually an evidence of the longitudinal variety of petrous fracture, except in those cases where the glenoid cavity has been shattered by a blow on the chin. In these cases we may get bleeding from an isolated fracture of the external auditory canal, with or without a ruptured drum. Mellenger[4] makes the following statement: "As a rule, transverse fractures of the pyramid do not involve the membrana tympani, therefore hemorrhage from the ear in head injuries usually means a longitudinal fracture of the pyramid and is almost pathognomonic of middle fossa fractures." Of course, this rule does not hold when the transverse fracture is combined with a longitudinal fracture, and even in the pure, uncomplicated labyrinthine fractures there have been a few exceptions to the rule. (Cases reported by Voss,[3] Biechele[26] and Schittler[38].)

The sources of the hemorrhage in these cases are fractures of the external canal, the tympanic plexus of veins, the sigmoid sinus, the superior petrosal sinus, the jugular bulb, the middle meningeal artery and, in a few rare cases, also the internal carotid artery. When the internal carotid artery has

been injured, the hemorrhage is cataclysmic in character and occurs at the same time from nose, mouth and ear. In these cases, death usually ensues very quickly.

In my series of 211 cases of skull fracture, 112 gave a history of having bled from one ear, and 34 from both ears. The unilateral hemorrhage was from the right ear in 65 cases, and from the left in 46 cases. Voss[3] states that he has never seen a case which bled from both ears, and Davis[45] reports that his mortality in those cases which bled from both ears was 66 per cent.

The initial bleeding which occurs after a skull fracture is well known, but the fact that hemorrhage from the ear can occur at a considerable time after the injury has, to my knowledge, been commented on only once in the literature. Forréstier[53] reported the case of a child who began to have a series of hemorrhages from the external canal three weeks after the accident, and this is somewhat analogous to a case reported by Vincenzo Palumbo[54] of an isolated fracture of the external canal, from which an escape of cerebrospinal fluid occurred three weeks after the injury and recurred intermittently for two years, at which time a meningitis developed. Hemorrhage into the labyrinth 17 days after a blow to the right parietal region was reported by Urbantschitsch[55] and this was evidenced by the sudden onset of tinnitus, deafness, nausea, vomiting and severe vertigo. The condition is also analogous to a cerebrospinal rhinorrhea which occurred three months after a fracture through the frontal sinuses reported by Cairns.[56] These late ear hemorrhages after skull fracture are also analogous to late hemorrhages into the brain, the occurrence of which after similar injuries has been recognized for many years.

At the January meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, I[57] reported seven cases in which late hemorrhage from the ear caused by head trauma occurred. In four of these cases the skull fracture was followed by immediate ear bleeding, which recurred a number of times after its initial cessation. In the other three cases, however, the first ear hemorrhage was reported one week after the injury in one case (Miss A. K.), and a month after the injury in the other two cases (R. N. and J. Q.) In four of these cases the skull fracture was confirmed by X-ray. We can only conjecture what the explanation of

these late hemorrhages from the ear may be. Nager[58] has never seen bleeding from the ear as late as my observations indicate, but supposes that it might be caused by a longitudinal fracture injuring the middle meningeal artery or one of its branches, and that it could not heal because of displacement of the fracture edges. The only cases of frequently repeated ear hemorrhages after head trauma which Voss[59] has seen have been due to injuries to the lateral sinus by a longitudinal fracture, and Brunner[60] suggests that they are analogous to the late hemorrhages into the brain and that they may possibly arise from traumatic aneurysms of small branches of the middle meningeal artery in the external canal, or from like aneurysms of small branches of the middle meningeal artery within the dura. The true explanation of these cases will, however, probably have to remain in the field of speculation until we have the opportunity to make a thorough histological study of the temporal bone and its adnexa in one of these cases.

The recurrence of the bleeding from time to time is not unlike the intermittent discharge of blood or cerebrospinal fluid from the nose, which is occasionally seen in cases of anterior fossa fracture.

HEMATOTYMPANUM.

There were seven cases in my series in which hematotympanum was observed. In hematotympanum the middle ear is filled with blood but the drum is not ruptured, and blood does not escape from the external canal. The drum itself exhibits more or less bulging and has a bluish or a bluish-red discoloration. This can only be seen on early inspection and is another reason why the otologist should see these cases early. Voss[3] found the condition eight times in his series of 104 cases. It probably occurs much more frequently than the eight cases of Voss and my own seven cases would indicate, because ordinarily the otologist does not see these cases early and the surgeon does not make a competent otoscopic examination.

Whereas, bleeding from the external canal is always the result of a longitudinal petrous fracture, and in a few rare cases of isolated fracture of the walls of the external canal, and is practically never the result of a labyrinthine fracture, hematotympanum can occur in either variety of fracture. In

the longitudinal variety it is found when the fracture has opened into the tympanic cavity but has not reached the margotympanicus or ruptured the drum. In the labyrinthine fracture it is seen when the fracture has involved the lateral wall of the internal ear. From the standpoint of diagnosis, therefore, the discovery of a hematotympanum does not indicate the type of petrous fracture present.

DISCHARGE OF CEREBROSPINAL FLUID AND LIQUOR TYMPANUM.

The escape of cerebrospinal fluid from the ear constitutes a definite sign of temporal bone fracture and indicates that the subarachnoid space has been opened by the fracture. It is not seen as frequently as bleeding from the ear. Besley[10] found it present in 2 per cent of 1,000 skull fractures. Gurdjian[46] found it only 11 times in 2,600 cases, while Voss[3] reported six instances among 66 cases of temporal bone fracture. Yerger[51] found it in 20 per cent of skull fractures. In my own series of 211 cases of skull fractures, the escape of cerebrospinal fluid was encountered eight times. (Cases 54, 71, 93, 149, 173, 175, 195, 210.) The discharge consists at first of blood mixed with spinal fluid, and the diagnosis of cerebrospinal fluid leakage could not be made with certainty until the second or third day, at a time when the bleeding tended to diminish. The discharge of cerebrospinal fluid usually ceases in a few days, but may continue for weeks, months and even years, and in one of my own cases continued for six weeks.

Like bleeding from the ear, the discharge of cerebrospinal fluid sometimes appears as late as 15 days after the fracture (Phelps,[52] Borden[48]). In one of my cases it did not appear until a week after the injury, and this was the case which continued to discharge fluid for six weeks (Case 54). Analogous conditions are observed in the post-traumatic cerebrospinal rhinorrheas and are explained by Duret[11] as due to the presence of inflammatory exudates which obliterate the osteomeningeal fissure until they are absorbed.

Voss'[3] opinion that all of the cases in which there is an escape of cerebrospinal fluid are due to labyrinthine fracture certainly cannot be corrobrated by my experience. Because of the fact that the cochlear and vestibular function was present in all of my cases, I would certainly classify them as longitudinal fractures. In none of them could I make the diagnosis

of inner ear fracture. Voss believes that these cases are all either transverse or combined transverse and longitudinal fractures. My series indicates that they are mostly longitudinal fractures, and that the escape of cerebrospinal fluid bears almost the same relation to the diagnosis of longitudinal fracture of the temporal bone as does bleeding from the ear.

Voss[3] has coined the term "liquor tympanum" for those cases in which cerebrospinal fluid is found in the tympanic cavity but the drum remains unruptured. In this condition, the drum may show some bulging but does not have the bluish appearance seen in hematotympanum. In one such case he observed a fluid level line, which remained horizontal in every position of the head. He states that Manasse and Koch have reported similar cases, and that Ruttin opened the mastoid in such a case and found the tympanic cavity and all the mastoid cells filled with liquor.

It is the opinion of Davis[45] that when bleeding from the ear is accompanied by the discharge of cerebrospinal fluid, the injury is more serious and all such cases have been fatal in his experience. Voss[3] and Schück[61] agree that the prognosis is more serious, and Linck regards it as an absolute indication for operation. On the other hand, Manasse, Brunner, Koch, B. Burger and Demmer (cited by Voss[3]) hold that the escape of cerebrospinal fluid does not aggravate the prognosis and is no indication for operation. In the light of my own experience, I must join this latter group. I have seen no fatalities in cases exhibiting a discharge of spinal fluid and none of these cases were operated upon. I have personally seen no case of liquor tympanum but the condition must exist and is quite analogous to hematotympanum.

LUMBAR PUNCTURE.

A lumbar puncture should be done on every case of serious head injury to determine whether the fluid is bloody or not. No harm can accrue from the removal of a small amount of spinal fluid and repeated punctures may prove very beneficial in relieving the increased intracranial tension. In the series of 1,000 cases reported by Wortis and Kennedy,[47] 846 had spinal punctures done and of these, 791 revealed a bloody spinal fluid. In a series of 224 acute head injuries, Temple Fay[62] had 104 cases with bloody spinal fluid, or 46.4 per cent,

and Gotten[63] reported that 65 per cent of his cases gave the same finding. Voss[3] believes that the findings of blood or a xanthochromic fluid points to a skull fracture, while Ramadier and Caussé[12] do not agree that it clinches the diagnosis and feel that it can be caused by brain injury without fracture. In this connection they quote Balthazard as having repeatedly shown in medicolegal autopsies the presence of hemorrhages in the meninges which were clearly traumatic without finding the least sign of cranial fracture.

Conversely, a skull fracture can occur without any bloody spinal fluid, as Stewart[64] was able to show in 10 per cent of the cases, and he believes that this figure would be considerably higher if one adds the cases of minimal fracture of the labyrinthine capsule. At any rate, the finding of bloody spinal fluid may be an indication that the skull has been fractured and surely aggravates the prognosis, for of 791 cases with bloody spinal fluid in the series of Wortis and Kennedy, 499 died.

ECCHYMOSES OVER THE MASTOID.

Ecchymosis developing slowly and progressively over the mastoid process is one of the classical signs of fracture of the base, and as a rule these ecchymoses in fractures of the temporal bone appear from the fourth to the fifth days following injury. They may also be observed in the external auditory canal. They have no connection with the hematoma frequently observed immediately after the injury at the point of impact and are seldom associated with bleeding from the ear but are, nevertheless, the result of a longitudinal fracture of the temporal bone. If the discoloration is very marked, one would be forced to suspect an injury to the lateral sinus. Not infrequently a suggilation about the eye is seen in connection with middle fossa fractures, and when this is present it points to an extension of the fracture into the anterior fossa. Voss[3] reports 10 such cases. In this connection, however, it must be borne in mind that suggilation and ecchymosis about the eye is a much more frequent finding in anterior fossa than in middle fossa fractures.

FACIAL PARALYSIS.

A facial paralysis occurred in 29 cases of my whole series, an incidence of 14 per cent. It was present in five cases in

which a diagnosis of some sort of a labyrinthine fracture had been made, therefore, in 31.25 per cent of these cases. It was present in 21 cases of longitudinal fracture, an incidence of about 18.7 per cent. The paralysis was complete and permanent in one transverse and in five longitudinal cases. In the remainder it either improved or cleared up completely. It appeared soon after the trauma in 16 cases; in one case (Case 9), four days after; in one case (Case 175), one week after; and in two cases (Cases 60 and 116), two weeks after the injury. In nine of the cases, the time when it first appeared could not be ascertained. A diagnosis of a petrous fracture was made in all but two of the cases developing facial paralysis, and in the latter such a type of fracture could not be excluded.

According to various authors, the incidence of facial paralysis varies greatly, but this discrepancy may, in some measure, be due to the accuracy of observation in various series. All authors agree that it is much more common in transverse than in longitudinal fractures, and my own observations would corroborate this viewpoint. According to statistics of Siebenmann, Brunner, Voss and Ulrich, transverse fractures cause a facial paralysis in about 50 per cent of the cases, and the longitudinal variety in from 10 to 18 per cent. In their series of 255 cases, Glaser and Schafer[65] found only 17 cases of facial paralysis, but many cases of their series of head injuries did not present skull fractures. Besley[10] found an involvement of the facial nerve recorded only 13 times in his 1,000 cases. Borden[48] found 24 cases of facial paralysis among 408 cases of skull fracture. On the other hand, Davis[45] reports a peripheral facial paralysis in 46 per cent of middle fossa fractures, and in the carefully studied series of Voss,[5] the incidence of facial nerve involvement was 18 per cent in longitudinal and 50 per cent in labyrinthine fractures, while Passow[16] reports facial paralysis in practically all fractures of the petrous pyramid. As my own records indicate, facial paralysis may appear either immediately or as late as 15 days after the injury.

Immediate paralysis is usually due to an actual lesion of the nerve by the line of fracture, either in the Fallopian canal or at the internal auditory meatus. It is usually complete. Such a lesion may be caused by a traction tearing, or actual

section of the nerve. In a few of these cases, the function of the nerve does not return. In the majority, the function either improves or returns completely after a variable period of time. This is due to the fact that the nerve is either simply stretched or that there takes place, according to Linck (cited by Ramadier and Caussé[13]), a regeneration of the nerve trunk, sectioned at the internal auditory meatus, or because the paralysis is due to an interfascicular hemorrhage, as many otologists contend.

Late facial paralysis appears during the first two weeks subsequent to the accident. It may be complete or incomplete, but, in contradistinction to the immediate form, cure is the rule, although it may require several months. Desmaulins (cited by Ramadier and Caussé[13]) and Ulrich[22] attribute this variety to a secondary neuritis of the nerve, either due to hemorrhage into the canal or accompanying the processes of repair. Cases of bilateral paralysis of the facial nerve in petrous bone fractures have been observed by Lannois and Jacod (cited by Ramadier and Caussé[13]), Mauthner[39] and Gurdjian.[16] Several of Mauthner's cases were cured.

RADIOGRAPHY.

Radiography can be a very valuable adjunct in the diagnosis of basal skull fracture, but the positive results of X-ray examinations depend very greatly upon the technique employed, the time at which the X-ray is taken, and the personal element. The Roentgenologists of most hospitals when called upon to ray a head for suspected skull fracture are content to take anteroposterior or lateral views, either in flat plates or stereoscopically, and while this technique is quite satisfactory for the delineation of vault fractures, it is wholly inadequate for basal skull fractures. Even when a satisfactory technique for fractures of the base is employed, the marked irregularities of the base, the varying degree of thickness and the multiplicity of foramina preclude a clear view of a fracture, particularly when it involves the middle cranial fossa.

According to the majority of the authors, the exposures should be taken in the positions described by Stenvers, Mayer, Schüller and Lange-Sonnenkalb. Using these views, Blohmke[66] believes that an absolute objective proof of basal skull frac-

ture can always be had. Gurdjian[46] reported 93 per cent positive X-ray findings in 379 cases of unilateral aural hemorrhage, and 95 per cent in cases of bilateral bleeding from the ear. The majority of authors, however, including Ramadier and Caussé[13], Voss,[3] Biechele,[26] Janke, Brunner[19] and Ulrich,[30] agree that the demonstration of a longitudinal fracture by X-ray is very difficult, and Lannois and Gaillard (cited by Voss), after reviewing the literature, X-raying skulls as well as living cases with fracture, conclude that only the most favorably placed basal fractures can be demonstrated by X-ray, whereas finer fissures and longitudinal fractures of the temporal bone cannot be demonstrated by X-ray.

The time element in the radiographic examination of the fractured skull is important. Fresh fractures show more easily than old ones, and if a middle fossa longitudinal fracture is suspected, the X-ray examination should be made early; *i.e.*, as soon as the condition of the patient warrants. Even then, and using all possible positions, a great many longitudinal fractures will be missed.

The story is quite different with the transverse fractures of the petrous pyramid. Here, because of the peculiarities in the bone regeneration of the capsule of the labyrinth, previously noted, fractures can be delineated by X-ray for a long time after injury. In these late X-rays, the fracture will not be as sharply defined as in early pictures because the periosteal and endosteal layers of bone near the periphery of the pyramid do show some tendency to close by bony union; but, nevertheless, they will be discernible. And for the determination of transverse fractures, the position of Stenvers is ideal. Lindemann[79] and Ulrich[30] in particular were able to demonstrate that these fractures appear on the X-ray plate for years after the injury. Ulrich called attention to certain differences between early and late X-rays. In the late X-rays, *1.* the fracture line was no longer visible on the sharp edge of the pyramid but only became apparent in the interior of the bone. *2.* The edges of the fracture line were no longer as sharp as originally. *3.* The fracture line was more distinctly visible in the interior of the petrous bone than at its periphery. He was still able to show the fracture line by X-ray two to 12 years after the fracture occurred.

In my series of 211 cases, X-ray was positive for skull fracture in 72 cases. In the remaining cases, radiological examination had either not been made or the findings were not obtainable, and in these the diagnosis was made from other clinical findings. In the majority of the 72 cases, the reports indicated vault fractures or vault fractures running into the base. In four cases a definite petrous bone fracture was delineated. In none of the cases of longitudinal fracture was the X-ray reported positive.

Under the heading of "Pathological Anatomy," I have already reported three cases of transverse fracture of the pyramid which I was able to demonstrate by X-ray three months, eight and one-half months and 11 months after the injury. In this connection, I would like to report a case in which the X-rays were positive eight years after the accident.

Case Report 157: Margaret M., age 13 years, who lived in a neighboring city, was injured in June, 1930, when she was knocked off of a bicycle to the pavement by a passing automobile. She presented herself for examination at my office on Oct. 8, 1930. The following history was obtained from her mother and her family physician. She was rendered unconscious for about four hours. The accident occurred at 6:00 P.M., and her physician was able to communicate with her the following morning and at that time she was able to hear him. By evening that same day she heard with great difficulty, and during the course of the following month she lost her hearing completely. X-rays taken at the hospital did not reveal any skull fractures. There was no bleeding from the mouth, nose or ears, but she vomited blood for 24 hours and at intervals thereafter. The girl had vertigo on the morning following the accident but her doctor did not consider this as violent in character. This was present even in the recumbent position but was aggravated by sitting up.

Past History: Scarlet fever one year preceding but with no sequellae relating to ears or hearing. No past history of otitis media or mastoid infection.

Present Complaint: Total deafness, vertigo in attacks when making sudden movements, tinnitus and occasional headaches.

Examination: No facial paralysis. Pupils equal and react promptly to light. Fundi negative. No ocular evidence of

increased intracranial tension. Vision normal. No evidence of fracture in either external canal. Both drums relatively negative. No scars; no perforations. Using a Barany noise apparatus in the left ear, a loud shout can be appreciated by the right ear but not interpreted. No hearing present in left ear. There was a spontaneous rotary horizontal nystagmus to each side in the end-position of the eyes. There was a spontaneous pastpointing to the right with the left arm. In forward and backward gait with eyes closed, she deviated first to one side and then to the other. The Romberg was negative. Turning tests: 10 turns to the right in 20 seconds produced no nystag-

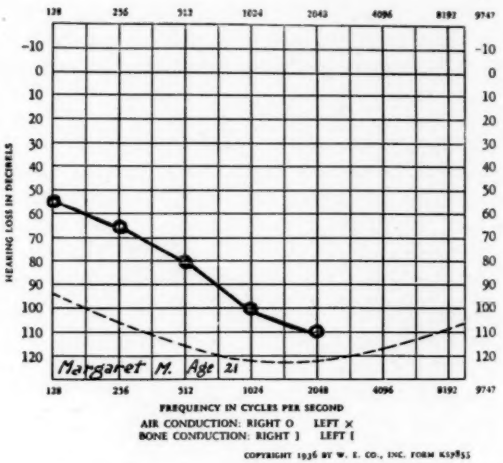

Fig. 8. Audiogram of Margaret M., Case 157.

mus; 10 turns to the left produced no nystagmus; 10 turns to the right in 10 seconds produced no pastpointing with the right arm, but a slight pastpointing to the right with the left arm. This corresponded to the spontaneous pastpointing above noted. Ten turns to the left produced no pastpointing with either arm; five slow turns to the right produced no falling reaction; nor did five turns to the left. Caloric irritability was not tested at this time.

Comment: From the results of this examination I believed that we were dealing with a bilateral transverse pyramid fracture.

On Oct. 22, 1938, I re-examined this patient, now 21 years of age. She could not hear speech but said she could hear music. She had learned to read lips and had completed a course as a beauty operator. The hearing for the voice was as before: no hearing with the left ear but a loud shout could be heard with the right. An examination of the right ear with the audiometer showed a subtotal loss of hearing. The left vestibular apparatus could not be stimulated by any amount of ice water. The right did not react until 90 cc. of ice water had been used, and then only gave a minimal

Fig. 9. X-ray (Stenver's position) showing transverse fracture of pyramid eight and one-half years after its occurrence.

response. X-rays in Stenver's position revealed an irregular fracture line across the left petrous bone near its base. but no fracture could be demonstrated on the right side.

Comment: This proves to be a case of transverse fracture of the left petrous pyramid with a total left cochleovestibular paralysis and an almost total loss of cochleovestibular function on the right side, probably due to massive hemorrhage

into the right labyrinth, although injury to the nerves at the internal auditory meatus cannot be excluded. The case also illustrates the fact that the petrous fracture can be delineated by X-ray eight and one-half years after it occurred.

DISTURBANCES IN COCHLEAR FUNCTION.

The functional examination for hearing is not reliable in the early days after the injury because of the disturbed condition of the sensorium. Most of these patients have suffered brain injury as well as fractures of the calvarium and one must delay the functional examination until the sensorium has sufficiently cleared; even then the results in individual cases are hard to evaluate. The examination of the hearing function is entirely subjective in character. We often have no knowledge of the hearing function of the individual before he was injured, and this is particularly true of older individuals. These cases are practically always medicolegal and one must be continually on guard for malingering. Reliable deductions can only be made after studying the findings of a large number of cases.

From the standpoint of the hearing function, my cases were separated into the following groups: *1.* Fractures involving various regions of the vault, face, sinuses and base, but apparently not involving the middle fossa or the temporal bone, 49 cases. *2.* Unilateral longitudinal fractures with bleeding from one ear, 112 cases. *3.* Bilateral longitudinal fractures with bleeding from both ears, 34 cases. *4.* Fractures of the petrous pyramid or labyrinthine fractures, pure or combined with longitudinal fractures, 16 cases. Each group was further subdivided into three groups according to age: *a.* 20 years and under; *b.* from 21 to 50 years, inclusive; and *c.* over 50 years of age.

The hearing function was studied by means of the voice, whisper, tuning forks and audiometer. Repeated examinations were made in most cases.

Inasmuch as it is impossible to give the results of individual hearing tests in so large a number of cases, the state of the hearing function was divided according to the audiometric findings into the following classes: *1.* normal; *2.* slight, if the hearing loss was less than 20 dcb.; *3.* moderate, when

the loss was between 20 and 40 dcb.; 4. marked, when between 40 and 80 dcb.; and 5. subtotal, when the hearing was further depreciated, but still present. I realize that this division is arbitrary but some such arrangement is necessary to study the relative effect of a particular lesion on the function of hearing in a large group of cases. A further attempt was made to diagnose the hearing loss as conductive, perceptive or combined in order to determine whether the seat of the lesion was in the middle ear or internal ear, but in the combined type of deafness this deduction must necessarily be inaccurate, for without pathological study of the individual case we cannot with absolute certainty say whether the loss for high tones is the result of a pure inner ear lesion or whether it may to some extent be the result of a middle ear lesion; this applies more particularly to the group of longitudinal fractures where definite damage to the middle ear function may be expected.

COCHLEAR FUNCTION IN SKULL FRACTURE OTHER THAN PETROUS BONE FRACTURES.

In this group there were 49 cases. The fracture was confined to the vault in 34, the vault and sinuses in one, the vault and base in seven, the anterior fossa in two, the base in two, the base and face in two, and the frontal sinus in one case. As far as I could determine, the petrous bone was spared in all.

In the age group of 20 and under, there were four cases and all the ears had normal hearing. In the next age group, from 21 to 50 years, inclusive, there were 37 cases. There was normal hearing or only slight loss in 36 ears. There was a moderate hearing loss in 17 ears, and in 12 of these it was of a perceptive type. There was a marked hearing defect in 16 ears, and in 15 of these the loss was of a perceptive or combined type. One ear showed a subtotal loss of hearing.

In the last age group (over 51 years) there were eight cases; in this group there were only two normal ears. Ten ears showed a moderate hearing loss, and four marked deafness.

In the entire number of 98 ears there were 45 which certainly gave some indication of inner ear damage. Even after

eliminating certain malingerers and some in whom the hearing defect was present before injury and, therefore, due to other causes, there certainly were many ears in which the defect in hearing was due to injury. In some the defect may have been caused by stretching of the cochlear nerve, occasioned by the side motion of the brain, as Ulrich contends, but we cannot deny that in many of these the defect was due to hemorrhage into the inner ear, which would place them in the category of commotio labyrinthi, a classification the existence of which many otologists deny; commotio labyrinthi will be discussed later.

TABLE II.
Cochlear Functions in Skull Fractures Not Involving the Petrous Bone.

Age	No. Cases	No. Ears	Hearing					Summary
			Slight Loss or Normal	Moderate Loss	Marked Loss	Subtotal Loss	Total Loss	
0-20	4	8	All normal	....	....	....	....	Indication of
21-50	37	74	36	17	16	1	....	perceptive
51+	8	16	2 normal	10	4	....	....	loss 45 ears

COCHLEAR FUNCTION IN UNILATERAL LONGITUDINAL FRACTURES.

This group consisted of 112 cases; in 10 of these the longitudinal fracture of the petrous bone was associated with fractures of other portions of the skull. Several cases were not included in this study of the hearing defects because the age of the patient was not known. The hearing was tested in 108 cases.

In the group of 20 years or under, 15 cases were studied. The ear of the side involved by fracture was normal or showed only slight hearing loss in seven, moderate hearing loss in four, and marked loss of hearing in four. On the side unaffected by the fracture, 11 were normal or showed only slight loss, three showed a moderate loss, and one showed a marked loss of hearing. In this group, both ears showed the same degree of involvement in five, the ear on the side of the fracture showed more marked involvement than its fellow in seven, and less involvement than its fellow in three cases. There was an inner ear type of hearing loss in 11 ears on the side of the fracture, and in five on the opposite side.

In the group of 21 to 50 years, inclusive, 82 cases were studied. The ear on the side of the fracture was normal or showed only slight hearing loss 17 times, moderate loss 24 times, marked hearing loss 18 times, and subtotal loss five times. The opposite ear was normal or showed only slight loss 35 times, moderate loss 26 times, marked loss 19 times; there were no ears with subtotal loss. In this group, both ears showed the same degree of involvement in 31, the ear on the side of the lesion was more involved than its fellow in 38, and was less involved than its fellow in six cases. There was probably inner ear involvement in 69 ears on the same side as the fracture, and in 52 ears of the opposite side.

In the group over 51 years there were 11 cases. None of the ears were normal and there were none showing slight involvement. The ear on the side of the fracture showed a moderate loss of hearing once, marked loss nine times, and a subtotal loss once. The opposite ear showed a moderate loss of hearing two times, a marked loss seven times, and no subtotal loss. All cases gave evidence of inner ear involvement.

In all three groups, 216 ears were studied. Discounting the ears with normal or only slightly impaired hearing, there was considerable loss of hearing of variable degree in 86 ears, or 79.5 per cent on the homolateral, and in 59 ears, or 54.6 per cent, on the contralateral side.

From this summary one can make several general deductions: *1.* The function of hearing is more severely damaged by the longitudinal fracture as age increases, the percentage of normal or slightly damaged ears being 60 per cent in the first group, 32.4 per cent in the second group, and nothing in the third group. *2.* Although the ear on the side of the fracture more frequently showed a greater loss of hearing than its fellow, still in many of the cases the degree of hearing loss was almost the same in both ears. This observation, taken in conjunction with the large number of ears which showed an inner ear type of deafness, indicates to me that we cannot discard the classification of commotio labyrinthi, as so many otologists would have us do, if by commotio we understand a damage to the end-organ by hemorrhage into the inner ear.

COCHLEAR FUNCTION IN BILATERAL LONGITUDINAL FRACTURES.

Thirty-four cases in which there was bleeding from both ears were studied; of these, six were age 20 years or under, 23 were between 21 and 50 years, inclusive, and five were over 50 years old. In the first group, eight ears were normal or showed only slight hearing loss, three showed a moderate conduction loss, and one a marked perception loss. In the next age group there were seven normal ears, eight with only slight loss, nine with a moderate loss, 20 with a marked loss,

TABLE III.
Cochlear Function in Longitudinal Fractures.

Type of Fracture	Age	No. Cases	No. Ears	Hearing: Slight Loss or Normal	Hearing: Moderate Loss	Hearing: Marked Loss	Hearing: Subtotal Loss	Hearing: Total Loss	Summary
Unilateral 112 cases	0-20	15	30	*H 7 *C 11	4	4	....	....	Same degree B 5 More marked H 7 More marked C 3 Perceptive H 11 Perceptive C 5
	21-50	82	164	H 17 C 35	24 26	18 19	5 0	....	Same degree B 31 More marked H 38 More marked C 6 Perceptive H 69 Perceptive C 52
	51+	11	22	H 0 C 0	1 2	9 7	1 0	....	Perceptive (all) Consid. loss: H. 79.5% C. 54.6%
Bilateral 34 cases	0-20	6	12	8	3	1	....	....	Indication of perceptive loss, 43 ears.
	21-50	23	46	7 normal 8 slight	9	20	2	....	
	51+	5	8	0	....	7	1	....	Consid. loss 65%

*(H) Homolateral, (C) contralateral, and (B) bilateral.

and two with subtotal loss of hearing. In the last group there were no ears with either normal hearing or only a slight loss, seven with a marked loss, and one with a subtotal loss of hearing. In one of the cases of this group, hearing tests were not made. In the first group there were two, in the second group, 33, and in the last group, eight ears which seemed to indicate inner ear involvement. In the 66 ears of cases with bilateral bleeding, there was considerable loss of hearing in 43 ears, or 65 per cent.

The remarkable feature of this whole group of bilateral longitudinal fractures was that there were 12 ears with normal hearing and 11 ears with only a slight hearing involvement. Here again, as is true in unilateral longitudinal fractures, the amount of the hearing defect increases with the age of the patient.

HEARING FUNCTION IN LABYRINTHINE FRACTURES.

In this group there were 16 cases; there was total deafness on the affected side in all the cases. On the contralateral side there were three ears with normal hearing, three with slightly affected hearing, three with a moderate loss, six with a marked loss, and one with a subtotal loss of hearing. There was apparently an inner ear type of deafness in 10 of these contralateral ears.

Voss[3] had five cases of total deafness in which he made the diagnosis of longitudinal fracture but is unable to say, without histological studies, whether the deafness was due to an isolated tearing of the acoustic nerve in the sense of Ulrich, an inner ear fracture not discernible by X-ray, an endolymphatic hemorrhage or a fluid wave of liquor. In his cases of longitudinal fracture he found a high grade deafness in 35.6 per cent, a moderate loss of hearing in 8 per cent, but he tested the hearing function with the whisper alone. Davis[45] states that most of the deafness he encountered in these cases was of a conductive type, that any improvement which takes place occurs in the first eight weeks, and that he has never seen a case of inner ear deafness caused either by concussion or by fracture of the middle fossa. Brunner[19] does not believe the old teaching that longitudinal fractures lead to deafness and states that such loss of hearing as he found was present in only 35 per cent of his cases and affected the inner ear only mildly.

My investigation of the hearing function in skull fractures would indicate that the ear is moderately or seriously damaged in 49 per cent of the fractures which do not involve the petrous pyramid directly, and that in 45.7 per cent the damage is of an inner ear type. The longitudinal fractures of the temporal bone cause a hearing loss in 63.4 per cent of the ears, and here also the damage is more often of an inner ear than

a middle ear type. In the group of labyrinthine fractures, the deafness is complete in all of the homolateral ears, and the hearing is considerably damaged in 65 per cent of the contralateral ears.

DISTURBANCES OF EQUILIBRIUM IN SKULL FRACTURES.

Dizziness is one of the most common complaints after injury to the head but, as I have previously pointed out, all forms of dizziness are not otitic in origin. A turning vertigo is a certain sign of vestibular disturbance. Errors of sensation, such as tilting of the bed, a rising of the floor, a falling of the ceiling or walls of a room, a feeling of uncertainty or of being drunk may be of vestibular origin if accompanied by a hyperirritability of the vestibular mechanism to caloric stimulation or a marked inequality of the two vestibular mechanisms to such stimulation. This is especially true when the complaint of vertigo is accompanied by spontaneous rotary-horizontal nystagmus, spontaneous pastpointing, spontaneous drift of the arms in the deviation test, and anomalies of gait or station.

When there is marked hemorrhage into the vestibule or when the vestibule has been fractured, the vertigo is violent, excessive and continuous, but tends to disappear after a few weeks. When the vertigo is not of this type but comes on in spells or attacks caused by sudden movements of the head, elevation above ground level, muscular effort, exposure to sun and heat, and even very mild indulgence in alcohol, it does not arise from the end-organ but is due to a disturbance in the central vestibular nuclear territory occasioned by hemorrhage, scarring and vasomotor disturbances.

In the group of skull fractures which did not directly involve the petrous bone, consisting of 49 cases, vertigo was complained of and objective vestibular disturbances were present in 37, or 75.5 per cent. In the group of unilateral longitudinal fractures, they were present in 82 out of 112 cases, or 73.2 per cent, and in the group of bilateral longitudinal fractures they were present in all but eight of the 34 cases, or 76.5 per cent. Only four of the 16 cases of labyrinthine fractures did not complain of vertigo. My observations also indicate that vestibular disturbances are more likely to occur as age increases.

COMMOTIO LABYRINTHI.

In 1905, Passow[16] classified temporal bone injuries into: *1.* fractures of the labyrinthine capsule; and *2.* hemorrhages into the labyrinth, or commotio labyrinthi. The researches of Stenger[9] and Brunner[18] on experimental animals, and the pathological findings of Barnick[7] Sakai,[20] Schönbauer and Brunner[8] and others show that hemorrhage into the inner ear can take place without any fracture of the labyrinthine capsule. Ramadier and Caussé,[13] Mauthner,[30] Mellenger[4] and others agree that a commotio labyrinthi without any damage to the osseous shell of the labyrinth can and does occur. Opposed to this view are Uffenorde,[33] Ulrich[17, 22] and Klingenberg.[34] I have found many cases of serious or profound hearing loss after head trauma in which careful investigation failed to reveal any evidence of skull fracture; in other words, cases of pure brain concussion. I have also found, as these records show, many cases of longitudinal fracture of the petrous bone in which there was a marked hearing loss, not only on the side of the fracture, but also in the opposite ear. To say that the hearing defect in all of these cases is due to a tearing or stretching of the acoustic nerve and not to hemorrhage into the inner ear, as Ulrich does, is begging the question, particularly in view of the pathological studies above noted. I must, therefore, join with those who believe that these changes in the hearing function are caused, at least in part, by hemorrhage into the inner ear or by commotio labyrinthi.

DIAGNOSIS.

The diagnosis of a longitudinal fracture will rest upon the presence or absence of bleeding from the ear, hematotympanum, the escape of cerebrospinal fluid, liquor tympanum, visible fractures of the external canal walls, a positive X-ray when obtainable, and the functional examination of the ear. The presence of bleeding or a hematotympanum makes the diagnosis of such a fracture very likely. Its absence, or the absence of a drum rupture does not exclude it. The escape of cerebrospinal fluid from the ear is definite evidence of a longitudinal fracture if some hearing remains, but its presence in the middle ear without a rupture of the drum may be due either to a longitudinal or a labyrinthine fracture. A positive X-ray is a great help for conclusive evidence, but a neg-

ative X-ray finding in no way excludes the fracture because, in my opinion, the depiction of a middle fossa fracture by X-ray is extremely difficult. The functional examination is important evidence.

The diagnosis of transverse fractures of the pyramid is based upon history, the X-ray, the otoscopic examination and the functional examination. The history will assist because in most of these fractures the trauma to the head is occipital or frontal. The X-ray is of much more assistance here than in the case of the longitudinal fracture, particularly if the fracture line is gross enough; fine fissures of the labyrinthine capsule are frequently not discernible. Furthermore, when once seen on an X-ray plate the fracture can again be found on subsequent radiographs for many years. The otoscopic examination reveals a normal external auditory canal and either a normal drum or a condition of liquor tympanum. Occasionally a hematotympanum without drum rupture is the result of a labyrinthine fracture which reaches the lateral labyrinthine wall. The functional examination is not reliable in the first few days after injury but will later reveal a complete cochleovestibular paralysis.

The diagnosis of a fracture involving only a portion of the labyrinth and destroying only one of its functions is not easy in the living patient. The X-ray may help; if the fissures are fine it may fail. We must never lose sight of the fact that the same clinical findings can result from a massive hemorrhage into one of the compartments of the inner ear or to an isolated injury of one or the other branch of the VIIIth nerve in the sense of Ulrich. In the living, this diagnosis can, therefore, only be made tentatively.

COMPLICATIONS AND PROGNOSIS.

The mortality of skull fractures in general according to various statistics varies from 10.6 per cent to 37.8 per cent. (Gotten,[63], 10.6 per cent; Fay,[62] 18.3 per cent; Swift,[68], 25.15 per cent; Schönbauer and Brunner,[8] in a series from the von Eiselsberg Clinic, 27.7 per cent; Wortis and Kennedy,[47] 37.8 per cent). Most of the deaths occur within the first 48 hours. Thus, Angelesco (cited by Ramadier and Caussé[13]) reported that 87 deaths out of 100 occurred in the first 48 hours. The

bulk of this early mortality is caused by shock, cerebral contusion, cerebral hypertension or intracranial hemorrhage. There remain approximately 15 cases per 100 in which death occurs later and in which meningitis is a predominant factor, although the patient may also die of prolonged shock or bronchopneumonia.

Considering basal skull fractures alone, the mortality rate assumes still more alarming proportions. Angelesco (cited by Voss[3]) reported 36 deaths in a series of 130 cases, 22 of which died in the first 48 hours. Schönbauer and Brunner,[8] combining the statistics of Chadowsky, A. Heer, Murney, Burkhardt, Frank, Crandon and Wilson, Graaf and Schaack, gave an average mortality of 52.9 per cent, and Grete,[69] reviewing the statistics of A. Heer, Graf, Valentin, Lange and Phelps, consisting all told of 486 cases, placed the mortality at 53.24 per cent. Davis[45] found a mortality of 39 per cent in cases with unilateral bleeding, and 66 per cent where the bleeding was from both ears, and Gurdjian's[46] figures in this respect are quite similar, being 38 per cent and 67 per cent, respectively.

In the cases which survive the first 48 hours, the most frequent cause of death is meningitis. Among these there were 26 in the 486 deaths studied by Grete. Borden[48] found meningitis in 8 per cent of 35 autopsies, Brunn (cited by Yerger) in 8 per cent of 470 cases, Moody[49] in 7 per cent of 547 cases, and Vance[70] found 48 cases of meningitis in 512 cases of basal skull fractures. These figures, however, include all basal skull fractures, those of the anterior as well as the middle fossa. Meningitis frequently occurs in the first few weeks following injury but it can occur later when the case has apparently made a successful convalescence. In fact, because of the peculiar healing properties of the bone of the labyrinthine capsule alluded to above, the individual who has suffered a transverse fracture of the pyramid is never free of this danger. The development of otitis media in such a case is a danger signal which must be carefully watched. In fact, when the roof of the Eustachian tube has been fractured along with the labyrinth meningitis can complicate the picture without the appearance of a frank otitis.

Longitudinal fractures offer some risk of endocranial complications if a middle ear infection soon follows the rupture of

the drum. The danger of intracranial complication subsequent to this type of fracture usually only exists if suppuration appears soon after the rupture of the drum. If it does not develop soon after a longitudinal fracture, there is very little risk of later meningeal infection. The union of the fracture, whether osseous, fibrous or fibro-osseous, apparently produces sufficient barrier to the passage of infection. Fraser, in the discussion of Nager's paper,[44] cited the case of a patient who had died of meningitis following an otitis media one year after a basal skull fracture, confirmed by microscopic examination. The fracture had involved the roof of the antrum but had completely spared the labyrinthine capsule, therefore being definitely of the longitudinal variety. This case illustrates that the translabyrinthine route is not the only one taken by a late meningitis after fracture of the temporal bone. To the best of my knowledge, this is the only observation of this sort in the literature, and I will now offer one of my own.

Case Report 169: Paul P., age 57 years, was injured on July 7, 1928. While loading a mortar box onto a truck, he fell backwards off the truck, striking his head on the pavement. He was rendered unconscious and remained so for several days. At the time of injury he was bleeding from nose, mouth and the right ear. He was sent to the Emergency Hospital, where he remained for one and one-half hours. While there, he had a convulsion. He was transferred to the Misericordia Hospital, where he remained for 24 days. While there, he had several convulsions. Upon recovering consciousness he had a mild mania and was delirious for about two weeks.

On July 25, 1928, his physician reported "hearing still very much impaired."

On Sept. 4, 1928, his physician reported "impacted cerumen removed from both ears, which improved his hearing very much. He still complains of pain in the head and dizzy attacks." At this time he complained of almost total deafness in the right ear.

He returned to work on Sept. 10, 1928, and continued to work until Nov. 24, 1928, when he was obliged to stop work because of severe headache. On this date, his physician reported a temperature of 103° F., pin point pupils and pro-

jectile vomiting. Later in the day there was a convulsion, although there had been no convulsions since his hospital discharge. He was again hospitalized. On the following day rigidity of the neck and a left-sided facial paralysis appeared. The spinal fluid was under pressure, showed a cell count of 3,017, and a pneumococcus present.

I saw this patient for the first time on Nov. 26, 1928. He was comatose and had a Cheyne-Stokes respiration. The fundi were negative. The left external canal and drum were negative. The right external canal was clear. There were no perforations of the drum. There was a fine leash of blood vessels coming over Shrapnell's membrane from the posterior superior canal wall, which was ridged and reddened (undoubtedly a fracture line).

He expired on Nov. 27, 1928; diagnosis: meningitis. No autopsy was obtainable.

Comment: I have classified this case as one of longitudinal fracture because of the bleeding from the ear and because of the fact that while the hearing of his right ear was markedly impaired it was not totally deaf. The possibility that the right labyrinth was also fissured by the fracture cannot be excluded, because no necropsy was obtainable. No X-ray information was available. This case also emphasizes the necessity of early otological examinations in head injuries. This case and that of Fraser are the only cases of longitudinal fracture of the petrous bone in which a late meningitis developed thus far reported in the literature.

That the appearance of ear suppuration shortly after a longitudinal fracture does not make the prognosis worse than it would be without the presence of the fracture is shown by several cases in this series. In Cases 103 and 127 the ear which bled discharged pus for several weeks and then cleared up. Case 167 suffered a bilateral longitudinal fracture with bleeding from both ears. The patient had at the time of injury a chronic otitis media on the left side. He was not operated upon. Case 22 suffered a bilateral longitudinal fracture, followed by right otitis media and subsequently by the development of a cholesteatoma. She is still living, five years after her injury, and operative interference has been refused. Case 85 developed an otitis media, followed by definite mas-

toid symptoms, which cleared up without operation. Cases 147 and 177 developed otitis media and mastoiditis after longitudinal fractures. The mastoids were operated upon, and both recovered. Case 177 is particularly interesting because during the simple mastoid operation a fracture line was encountered, running across the upper portion of the mastoid toward the attic.

These cases would indicate that the appearance of a suppurative process in the ear which has bled following a basal skull fracture does not make for a worse prognosis than it would occasion by itself and, in my opinion, the same rules should govern therapy as would be laid down for the treatment of the same suppuration uncomplicated by the longitudinal fracture.

The nine cases in my series of 211 skull fractures which discharged cerebrospinal fluid from the ear for a variable period, plus a similar case in my shorter series, all of which recovered without any suppurative complications of the ear, controvert the absolutely black prognosis given to this symptom by Davis,[45] who stated that he had seen four cases in which the escape of cerebrospinal fluid accompanied the bleeding from the ear, and all four had been fatal. I do not regard the escape of cerebrospinal fluid as making the prognosis any worse than the escape of blood from the ear as long as the functional tests indicate no labyrinthine fracture.

The prognosis of transverse fractures, and particularly of combined transverse and longitudinal fractures, as to life is definitely worse than that for pure longitudinal fractures, inasmuch as the danger of late traumatic meningitis is considerably greater. This concerns, naturally, only those cases in which the fracture has reached the lateral wall of the inner ear. If this wall is spared, which is relatively seldom, then the danger of infection from the tube and the middle ear is gone. The danger of a late meningitis in these cases is due to the poor healing qualities of certain portions of the labyrinthine shell alluded to above. These fractures cicatrize in such a way that a pathway for bacteria is left indefinitely. If, under these circumstances, even a mild otitis appears, years after the injury, there is a great chance of its being complicated by a labyrinth infection and meningitis. The

length of time intervening between the fracture and the meningitis is illustrated by the following case reports listed in the literature: Scheibe, three weeks; Scheibe, 28 days; M. Meyer,[71] four weeks; Politzer,[44] 40 days; Politzer,[44] 50 days; Klestadt,[44] 50 days; Nager,[44] six months, Nager,[44] 210 days; R. Hoffmann,[44] 209 days; Ruyter,[13] seven months; Ramadier,[13] 15 months; Brock,[72] 15 years; and Schittler,[13] 16 years. These cases were all checked by microscopic study. An excellent review of this particular subject has been presented by Nager.[43, 44] I have not seen late meningitis in my series of 16 cases tentatively diagnosed as labyrinthine fracture.

Brain abscess may occur after a basal skull fracture, but the literature reveals that this complication is quite rare. In 1932, Grete,[69] of Nager's Clinic, reported the case of a man, age 37 years, who sustained a basal fracture involving the occipital and left parietal bones. The first otological examination on the fifteenth day revealed suppuration of the right ear. Three and one-half months later there were symptoms of brain abscess, and operation revealed a temporal lobe abscess. Grete also reports a case of Beck of a bilateral longitudinal fracture in which suppuration appeared in the left ear, and exitus from meningitis occurred five weeks after the accident. Autopsy revealed a diffuse purulent meningitis and a deep-seated abscess in the left temporal lobe. In 1915, Ruttin (Grete[69]) reported a case of longitudinal fracture in which death occurred two months after the accident, and the autopsy revealed a large abscess in the right temporal lobe and a fracture through the squama to the petrous bone.

Dixon[73] reported a very interesting complication in the case of a male, age 27 years, who had been thrown from a car to the pavement and brought unconscious to the hospital. X-rays revealed a stellate fracture of the posterior portion of the parietal and occipital bones on the left side. There was no bleeding from either ear but when first seen by Dixon on the fifth day there was a hematotympanum on the left side. The temperature was normal up to the fifth day and then rose to 101°. At this time pain developed in the left ear and over the course of the left jugular vein. On the following day there was a chill and the temperature rose to 104.2°. Twenty-four hours later there was another chill. Operation revealed a

normal mastoid. Near the upper end of the sigmoid sinus there was an old blood clot. The sinus was completely thrombosed; its wall was smooth and presented no visible tear. Recovery ensued.

A still rarer complication of a bilateral longitudinal fracture is an injury of either one of the petrosal sinuses or of the cavernous sinus, of which the following case report is an example.

Case 203: Joseph K., age 27 years, was injured on May 25, 1936. His head was crushed between two boxcars, and he was obliged to stand there until the two cars were pulled apart. There was no unconsciousness at this time. He was removed to the hospital and upon arriving there he became unconscious. Thereafter he was either unconscious or delirious for two and one-half months. Information from his attending physician reveals the fact that he was bleeding from nose, mouth and both ears, more profusely on the left side. On Sept. 12, 1936, his physician ligated the external carotid artery on the left side, presumably because of a proptosis and pulsating exophthalmos of the left eye. A bruit returned after this operation, and on Dec. 7, 1937, the left internal carotid artery was tied off. His face became crooked but he does not remember when this occurred. Since his accident he has had a numbness in the left side of his face and of the roof of his mouth. He apparently also had a partial paralysis of the left arm and left leg, but whether this came on after the accident or followed one of the operations it is difficult to elicit. No X-ray information was obtainable.

When examined on June 23, 1938, at the Columbia Hospital and at my office, his complaints were the following: *1.* a bulging left eye; *2.* a bruit every morning and at night, which was continuous in character until he had his second operation; *3.* his face is crooked; *4.* numbness of the left side of his face and the whole of his mouth; *5.* disturbed sense of taste. My examintion revealed the following: There was a complete left facial paralysis, with contractures. There was a marked proptosis of the left eye, associated with limitation of its movements, a pseudo-ptosis and a vision reduced to 20/100. His hearing was normal for voice, tuning forks and audiometer. He offered no complaint of vertigo, and objec-

tive examination of his vestibular mechanism was normal. X-rays of his skull taken at this time were negative for fracture.

Comment: The interesting features of this case were the bilateral longitudinal fracture of the petrous bones, evidenced by bleeding from both ears, the absence of immediate unconsciousness, followed by a long period of unconsciousness and delirium, the complete facial paralysis, the pulsating exophthalmos, associated with a bruit. This was caused by an arteriovenous aneurysm of aseptic character between internal carotid artery and cavernous sinus, a traumatic condition to which I drew attention in 1936.[74]

THERAPY.

The therapy of skull fractures in general has undergone a marked change in the United States during the last 15 to 20 years. The open operations of the trephine and subtemporal decompressions formerly practiced on all head injuries showing signs of increased intracranial tension are now reserved for such specific conditions as depressed fractures, extradural hematomata and middle meningeal hemorrhage. The present-day treatment for the control of increased intracranial tension consists in the limitation of fluid intake, the use of various hypertonic solutions by the intravenous route, free catharsis and repeated lumbar punctures. This has resulted in a marked reduction of the mortality in this class of cases, if the statistics of Fay,[62] Gotten[63] and others are any indication.

In the special otological treatment of the ear which is bleeding, one should be content to cover the ear with sterile dressings and not handle it in any other way unless suppuration ensues. I certainly cannot recommend the treatment of Ramadier,[13] Davis[45] and others, of cleansing the external canal with alcohol or peroxide and placing an iodoform wick in the canal. If the bleeding is so profuse as to lead one to the conclusion that the sigmoid sinus has been injured, one may have to expose and block the sinus.

In the treatment of fractures of the temporal bone, the classical rule has been to avoid operative interference unless a suppuration occurred. At the present time opinions on this

point are divided. Voss,[3] and more particularly Linck (cited by Voss), advocate active surgery in all types of temporal bone fractures. The majority of otologists, however, are inclined to be much more conservative.

In longitudinal fractures associated with bleeding from the ear, where no suppurative process existed in the ear prior to the fracture and none sets in, most otologists, with the possible exception of Linck, are in agreement that an operation is indefensible. If an otitis media and a mastoid suppuration develops, Ramadier and Caussé[13] advise operating, preferably in the radical manner. Inasmuch as he has succeeded very well by conservative methods in 17 cases of severe basal skull fractures with otitis media, Brunner[19] would individualize these cases, and draws a sharp distinction between the longitudinal and the transverse types of fractures. In longitudinal fractures complicated by otitic suppuration, he only advises surgery if the infection has extended beyond the middle ear (presence of fever, chills, headache, mastoiditis, meningeal symptoms, etc.). Harrison[75] operated on such a case 16 days after the fracture, with complete recovery. With a pre-existing otitis media, Nager[43] would not operate unless signs of meningeal irritation appeared. Fraser[76] did operate upon three such cases, and all of them died. If suppuration supervenes in a bleeding ear, Davis[45] does a simple mastoid operation. Voss[3] operates in all secondary infections in the neighborhood of the middle ear or mastoid. Ulrich[22] and Biechele[26] are inclined to treat these cases conservatively. Loebell,[77] on the other hand, believes that surgical intervention becomes imperative when a suppurative process develops in a traumatically damaged ear, or in case the cerebrospinal fluid or bloody discharge suddenly becomes infected, and in cases of fracture through an already suppurative ear, particularly if the pre-existing suppurative process flares up. My own experience with six cases in which suppuration followed the fracture and one case in which the fracture occurred through an already existing suppuration convinces me that such suppuration is not ordinarily complicated by endocranial invasion, at least to no greater degree than would exist in the suppurative process itself if uncomplicated by a fracture. The indications for operative procedure are those of the suppurative process itself. I have operated upon two of these cases, but only because the suppuration as such required the operation. Nor

does the spectre of a late ear infection, followed by meningitis, rear its head in this type of case, for in spite of the countless number of cases of longitudinal fractures of the petrous bone, associated with a ruptured membrana tympani and bleeding from the ear, only two cases of late ear suppuration terminating in meningitis have thus far been reported, one by Fraser and one by myself.

The question of operative interference in labyrinthine fractures is somewhat different, and this question is bound up with the fact that in approximately 50 per cent of these fractures the lateral labyrinthine wall is damaged and the labyrinth is put into communication with the middle ear. Inasmuch as microscopic examination has shown us that these fractures heal by fibrous tissue and not by bony union, the danger of a late meningitis always exists. Linck and Voss would operate on all labyrinthine fracture cases, infected or not, and they advise the radical mastoid operation, plus some form of labyrinth operation. Brunner[19] considers the situation very serious when an otitis follows a transverse fracture and, while a few of these cases have recovered under conservative treatment, believes that they should be operated upon. Nager,[43] summarizing the opinions of Manasse, Lange, Zange, Otto Mayer, etc., believes that the appearance of any infectious process in the organ of hearing after a transverse fracture is an absolute indication for operative exposure of the territory, and to this Ramadier and Caussé[13] state that the appearance of clinical symptoms of meningitis calls for an immediate radical operation, and if a labyrinthine fracture is found, also a labyrinth operation.

In my own experience with 16 cases of supposed labyrinth fracture, no ear suppuration has supervened to date, and I have advised no operative interference; however, because of the fact that in approximately half of these the internal wall of the middle ear has probably been fractured, I would advise careful clinical observation. Should an otitis media appear at any time, I would operate immediately, without waiting for an extension of the infection to the mastoid, or for the clinical signs of meningeal irritation. I would counsel an immediate radical mastoidectomy, with some form of a labyrinthine operation. Naturally, with the concomitant administration

of sulfanilamide the prognosis of such a case should be materially enhanced.

SUMMARY.

1. From an otological standpoint a clinical study of 211 skull fractures is presented. Of these, 49 were miscellaneous fractures of vault, face and sinuses, 146 longitudinal fractures of the temporal bone, and 16 fractures of the petrous pyramid.

2. The course of temporal bone fractures, their pathology, mode of production and repair processes are described.

3. Three cases of probable isolated cochlear fracture are presented.

4. The peculiarities of reparative processes in the labyrinthine capsule and the relation of these peculiarities in bone repair to the late demonstration of such fractures by X-ray are discussed.

5. For the diagnosis of temporal bone fractures, a careful evaluation of symptoms and signs is necessary. These consist of history of unconsciousness, early otoscopic examination, discharge of blood or cerebrospinal fluid, presence of hematotympanum or liquor tympanum, ecchymosis over the mastoid, facial paralysis, radiological examination and functional testing.

6. Seven cases of late hemorrhage from the ear after basal skull fractures are presented.

7. The escape of cerebrospinal fluid was encountered eight times. This sign indicates that the subarachnoid space has been opened, and in my experience has always been associated with longitudinal fractures. Also, from my experience the prognosis is relatively good both as to function and as to life.

8. Facial paralysis occurred in 31.25 per cent of the cases of petrous bone fracture, and in 18.7 per cent of the longitudinal fractures. It was complete and permanent in 20.7 per cent of the cases. The remainder recovered.

9. The X-ray is undependable in longitudinal fractures but very reliable in fractures of the otic capsule.

10. The examination of cochlear function is unreliable in the early weeks because of disturbed sensorium and other factors. In the group of miscellaneous skull and face fractures there was indication of perceptive deafness in 50 per cent of all the ears. In the unilateral longitudinal fracture group, the majority of ears showed a perceptive type of involvement, and though the ear on the side of the fracture frequently showed greater loss of hearing than its fellow, still in many cases the degree of hearing loss was the same on both sides. In the bilateral longitudinal fracture group, 65 per cent of the ears showed a considerable hearing defect, mostly of a perceptive type. In the transverse fracture cases, 56.2 per cent of the contralateral ears showed a considerable hearing loss. In all groups, the age factor is important, the loss of function increasing with the age of the patient.

11. Objective vestibular disturbances were found in the neighborhood of 75 per cent of all cases, irrespective of the type of fracture, and the origin of these was felt to be central rather than peripheral in the majority of cases.

12. Prognosis: Fracture of the otic capsule always presents the danger of a late meningitis even after many years. After the initial convalescence, the longitudinal fracture presents very little danger of later intracranial invasion.

13. The author cannot subscribe to the radical views on surgical intervention promulgated by Linck, Voss and others, and contents himself with a more conservative form of treatment; however, in the fractures through the otic capsule, subsequent otitic infection and suppuration must be closely watched, vigorously dealt with, and the labyrinth operation must be done on the earliest approach of meningeal symptoms.

ADDENDA.

Since writing the above, an extensive article on the hearing defects and disturbances of equilibrium caused by skull fractures, by A. Alexander and R. Scholl[80] has appeared and it seemed unwise to close this paper without making reference to their findings. Their material consisted of 551 cases of head trauma, only 387 of which they were able to follow up. They classified their 387 cases as follows: Commotio cerebri,

231 cases; contusio cerebri, 27 cases; face fractures, 22 cases; vault fractures, 40 cases; and base fractures, 67 cases.

They found objective evidence of hearing or vestibular damage in 10 per cent of the 387 cases. The disturbances of the vestibular apparatus were more numerous than those of the cochlear division. In the commotio cases with hearing complaints, a damage to the cochlear apparatus could only be demonstrated in one-seventh of the cases. In the cases of basal skull fracture with complaints of loss of hearing, damage to the cochlear apparatus could be demonstrated in 80 per cent.

With regard to the cochlear damage, they state that, whereas in general the more severe skull injuries more frequently cause hearing disturbances than the milder skull injuries, they are in full agreement with Koch, Voss, Uffenorde, Wittmaack and my own findings, that the mildest form of trauma may cause the most severe functional disturbance, and vice versa. They are also in agreement with various authors, that in general the degree of hearing defect depends upon the distance of the applied force from the ear.

They also call attention to the fact that cases in which the middle ear is damaged show an initial marked impairment of hearing which subsequently improves to considerable degree. Sixty per cent of their cases with hearing defects indicated a pure perceptive type of loss, unilateral more frequently than bilateral. The percentage of objectively demonstrated hearing defects increases with the severity of the head trauma, from 15 per cent in commotio cerebri to 80 per cent in basal skull fractures.

My general criticisms of this excellent survey are as follows: *1.* No mention is made of the method of conducting hearing examinations; if audiometric studies were not made, many minor hearing defects, especially of the perceptive type, were missed; *2.* their deductions on vestibular damage are based upon hyperexcitability, hypoexcitability and absence of reaction; they make no mention of a difference in reaction between the two sides; in other words, an imbalance; *3.* their deductions are all based upon late examinations made from one to six years after the injury and, therefore, discount all such vestibular injuries as may have become compensated dur-

ing the course of the first year. Their findings, however, fall within the scope of the title of their paper.

BIBLIOGRAPHY.

1. MYGIND, S. H.: Traumatic Vestibular Diseases. *Acta Oto-laryngol.*, 1:515, 1919.

2. GROVE, W. E.: Otological Observations in Trauma of the Head. *Arch. Otolaryngol.*, 8:249, 1928.

3. VOSS, OTTO: Die Chirurgie der Schädelbasisfrakturen. Johann Ambrosius Barth, Leipzig, 1936.

4. MELLENGER, W. J.: The Base of the Skull, with Particular Reference to Fractures. *Ann Otol., Rhinol. and Laryngol.*, 47:291, 1938.

5. RAWLINGS, L. BATHE: Head Injuries. Oxford Univ. Press, London, 1934.

6. BRUNS: Cited by Passow and by Biechele.

7. BARNICK, OTTO: Ueber Brüche des Schädelgrundes und die durch sie bedingten Blutungen in das Ohrlabyrinth. *Arch. f. Ohrenheilk.*, 43:23, 1897.

8. SCHONBAUER and BRUNNER: Schädelbasis Brüche; in Alexander and Marburg: Neurologie des Ohres, Vol. 2, 1927.

9. STENGER: Beitrag zur Kenntniss der nach Kopfverletzungen auftretenden Veränderungen im inneren Ohr. *Arch. f. Ohrenheilk.*, 79:43, 1909.

10. BESLEY, FREDERICK: A Contribution to the Study of Skull Fractures. *Jour. A. M. A.*, 66:345, 1932.

11. DURET: Cited by Ramadier and Caussé.

12. POULAIN: Cited by Ramadier and Caussé.

13. RAMADIER and CAUSSE: Traumatismes de l'Oreille. Masson and Cie, Paris, 1937.

14. STEWART: Cited by Ramadier and Caussé.

15. EHRENFRIED: Zur Kasuistik der Otitischen Meningitis durch Trauma. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 6:404, 1923.

16. PASSOW, A.: Die Verletzungen des Gehörorgans. Wiesbaden, 1905.

17. ULRICH, K.: Klinische und anatomische Untersuchungen über Verletzungen des Gehörorgans bei Schädelbasisfrakturen. *Schweiz. med. Wchnschr.*, 1921.

18. BRUNNER, H.: Pathologie u. Klinik der Erkrankungen des inneren Ohres nach stumpfen Schädeltraumen. *Monatsschr. f. Ohrenheilk.*, 59:697, 1925.

19. BRUNNER, H.: Ohr und Schädeltrauma. *Wien. klin. Wchnschr.*, 47:114, 1934.

20. SAKAI, K.: Anatomische Befunde am menschlichen Gehörorgan nach Basisfraktur. *Arch. f. Ohrenheilk.*, 85:188, 1911.

21. LANGE, W.: Beiträge zur pathologischen Anatomie der Verletzungen des Gehörorgans. *Passow u. Schaeffer Beitr.*, 18:277, 1922.

22. ULRICH, K.: Verletzungen des Gehörorgans bei Schädelbasisfrakturen. Suppl. to *Acta Oto-laryngol.*, 6:1, 1926.

23. THEODORE, E.: Beitrag zur Pathologie der Labyrintherschütterung. *Arch. f. Ohrenheilk.*, 61:299, 1910.

24. ALEXANDER, G.: Die Histologie der inderekten traumatischen Labyrinthverletzung durch Schädelschuss. *Festschr. f. Urbantschitsch*, 1918.

25. MANASSE, P.: Zur pathologischen Anatomie der traumatischen Taubheit. *Virchow's Arch. f. Path. Anat.*, 189:188, 1907.

26. BIECHELE, H.: Ueber die Mitbeteiligung des Felsenbeins bei Schädelgrundbrüchen. *Ztschr. f. Laryngol.*, 24:293, 1933.

27. GUERINOT: Cited by Ramadier and Caussé.

28. PROUST: Cited by Ramadier and Caussé.

29. FELIZET: Cited by Ramadier and Caussé.

30. ULRICH, K.: Heilungsvorgänge bei alten Labyrinthbrüchen in röntgenographischer Darstellung. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 35:263, 1934.

31. NAGER, F. R.: Ueber Spätmeningitis nach Labyrinthfraktur. *Acta Otolaryngol.*, 14:127, 1930.

32. HALPHEN: Cited by Ramadier and Caussé.

33. UFFENORDE, W.: Constatations histologique—Contribution a' l'etude de la surdité par commotion. *Beiträg. Anat. Phys. Path. des Ohres*, pp. 292-324, 1924.

34. KLINGENBERG, A.: Die isolierte Schneckenfraktur bei Schädelbasisbrüchen. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 22:452, 1929.

35. POLITZER, A.: Cited by Klingenberg.

36. DE KLEYN and STENVERS: Cited by Klingenberg.

37. HOFFMANN, LOTHAR: Cited by Klingenberg.

38. SCHITTLER, E.: Labyrinthzersplitterung — isolierte Vestibularfraktur — Spätmeningitis nach 16 Jahren. *Acta Oto-laryngol.*, 24:213, 1936.

39. MAUTHNER, O.: Die traumatische Erkrankungen des inneren Ohres. *Arch. f. Ohrenheilk.*, 87:146, 1911.

40. BROCK: Längsfraktur durch die Schläfenbeinpyramide mit verletzungen des inneren Ohres. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 34:349, 1933.

41. YOSHII, U.: Experimental Study of Traumatic Injuries of the Ear. *Internat. Zentralbl. f. Ohrenheilk.*, 9:561, 1910-1911.

42. KUNDRADT: Cited by Manasse.

43. NAGER, F. R.: Ueber Spätmeningitis nach Labyrinthfraktur. *Arch. f. Ohrenheilk.*, 122:217, 1929.

44. NAGER, F. R.: Ueber Spätmeningitis nach Labyrinthfraktur. *Acta Otolaryngol.*, 14:127, 1930.

45. DAVIS, E. D.: Injuries of the Ear Arising from Fractures of the Skull. *Brit. Med. Jour.*, II:741, 1928.

46. GURDJIAN, E. S.: Ear Complications in Acute Craniocerebral Injuries. *Radiol.*, 18:74, 1932.

47. WORTIS, S. B., and KENNEDY, F.: Acute Head Injury. *Surg., Gyn. and Obstet.*, 55:365, 1932.

48. BORDEN, CHAS. R.: Otological and Rhinological Complications of Skull Fractures. *Jour. A. M. A.*, 53:429, 1909.

49. MOODY: Cited by Yerger.

50. RANSAHOFF: Cited by Yerger.

51. YERGER, C. F.: Otological Complications of Basal Skull Fractures. *Arch. Otolaryngol.*, 4:35, 1926.

52. PHELPS, CHAS.: An Analytical and Statistical Review of 1,000 Cases of Head Injury. *Ann. Surg.*, 49:449, 1902.

53. FORRESTIER: Cited by Ramadier and Caussé.

54. PALUMBO: Cited by Ramadier and Caussé.

55. URBANTSCHITSCH, E.: Bericht. d. Oster. otol. Gesellsch. in *Monatsschr. f. Ohrenheilk. u. Laryngol.-Rhinol.*, 57:242, 1923.

56. CAIRNS, H.: Injuries of the Frontal and Ethmoid Sinuses. *Jour. Laryngol. and Otol.*, 52:589, 1937.

57. GROVE, W. E.: Late Ear Hemorrhage Following Skull Fractures. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, 1939.

58. NAGER, F.: Personal Communication.

59. VOSS, O.: Personal Communication.

60. BRUNNER, H.: Personal Communication.

61. SCHUCK, F.: Kopfverletzungen. Bericht über 300 Fälle. *Arch. klin. Chir.*, 153:77-93. Cited by Voss.

62. FAY, TEMPLE: Economic Readjustment Following Head Injuries. *Surg., Gyn. and Obstet.*, 54:362, 1932.

63. GOTTEN, NICHOLAS: Head Trauma. Report of 141 Cases. *Jour. A. M. A.*, 110:1727, 1938.

64. STEWART, I. P.: Fracture of the Temporal Bone, etc. *Jour. Laryngol. and Otol.*, 44:771, 1929.

65. GLASER, M. A., and SCHAFER, F. P.: Skull and Brain Traumas. Their Sequelae. *Jour. A. M. A.*, 98:271, 1932.

66. BLOHMKE: Die Bedeutung der Röntgen — Zielaufnahmen des Schläfenbeins, etc. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 17:311 and 18:173. Cited by Voss.

67. GROVE, W. E.: The Symptom of Vertigo. *Arch. Otolaryngol.*, 14:177, 1931.

68. SWIFT, GEORGE: Cerebral Injuries Due to External Trauma. *Surg., Gyn. and Obstet.*, 62:340, 1936.

69. GRETE, WALTER: Geheilter Temporallappenabszess nach Felsenbeinfraktur. *Arch. f. Ohrenheilk.*, etc., 131:245, 1932.

70. VANCE, B. M.: Fractures of the Skull. Complications and Causes of Death. *Arch. Surg.*, 14:1023, 1927.

71. MEYER, M.: Ausergewöhnliche Entstehung einer otogenen Meningitis durch Trauma, etc. *Ztschr. f. Laryngol.*, etc., 18:210, 1929.

72. BROCK: Spätmeningitis nach Labyrinthfraktur. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 34:360, 1933.

73. DIXON, O. J.: Thrombosis of the Sigmoid Sinus Following Skull Fracture. *Arch. Otolaryngol.*, 3:57, 1926.

74. GROVE, W. E.: Septic and Aseptic Types of Thrombosis of the Cavernous Sinus. *Arch. Otolaryngol.*, 24:29, 1936.

75. HARRISON, W. J.: Fractures of the Skull and Other Injuries, Followed by Acute Mastoid Disease. *Jour. Laryngol.*, etc., 43:342, 1928.

76. FRASER: In Discussion to Nager's Paper.[43]

77. LOEBELL, H.: *Ztschr. f. Laryngol.*, etc., 26:117, 1935.

78. BAST, T. H.: Personal Communication.

79. LINDEMANN, ERNA: Röntgenologische Untersuchungen über die Heilung von Schädelbrüchen. *Arch. f. Ohrenheilk.*, 135:25, 1933.

80. ALEXANDER, A., and SCHOLL, R.: Beschwerden und Störungen im Hör- und Gleichgewichts organ bei der Nachuntersuchung Schädelverletzter. *Monatsschr. f. Ohrenheilk.*, etc., 72:1021, 1938.

324 East Wisconsin Avenue.

REPORT OF THE CHICAGO COMMITTEE ON OTOGENIC MENINGITIS, 1939.*

DR. ALFRED LEWY, Chicago.

This year, the Chicago Committee planned to report only on pneumococcus meningitis. Seven unpublished authentic cases are known to us, with recovery. There may be a greater reluctance than formerly on the part of our colleagues to report their fatal cases since modern chemotherapy has so favorably influenced the results of treatment. At the time of this writing I have not received detailed reports of four of the above-mentioned cases which were promised me, and have included in the report one of streptococcus viridans, because of its rarity, and one in which the culture from the operated mastoid was reported as streptococcus hemolyticus, while the culture from the spinal fluid showed a Gram positive diplococcus. This I believe to be a mutation form, which, if true, only adds to the complexity of our bacteriological problem.

Case 1: White male, age 1 year, admitted to County Hospital, service of Dr. A. Levinson, Dec. 6, 1938, with a history of fever and vomiting of three days' duration. Temperature 104°; pharynx red; questionable loss of breath sounds in left base. Spinal puncture on admission showed five leukocytes per cmm., fluid clear, under slightly increased pressure; negative Pandy. Within two days patient developed bronchopneumonia and bilateral otitis media. Dec. 13, apathy and stiff neck; Dec. 15, bilateral mastoidectomy. Spinal puncture at this time showed cloudy fluid under pressure, 12,800 cells per cmm., mainly polymorphonuclears; glucose absent. The culture revealed a type IV pneumococcus, blood culture also showed type IV pneumococcus. Patient was given 80,000 units type IV pneumococcus rabbit serum, and sulfapyridine gr. 15 every four hours. The following day the dosage was doubled. Clinical improvement in 24 hours. By Dec. 22, spinal cultures were negative. The patient seemed fully recovered by Dec. 25. Sulfapyridine concentration in the blood was 2.8 mils. per 100 cc.

The child was discharged, Jan. 19, 1939, apparently totally deaf, but otherwise well. There has since been noted some questionable return of hearing.

Case 2: Male, age 35 years; diagnosis: asthma, hyperplastic sphenoethmoiditis, service of Dr. Noah Fox. Bilateral intranasal ethmoidectomy; following day, meningitis, 5,000 cells, culture pneumococcus type XVIII. Sulfapyridine gr. 15 every four hours; rabbit pneumococcus serum type XVIII given partly intraspinally, partly intravenously, also air injections intraspinally, as used in Vienna to prevent spinal block. Blood transfusions. Operation, March 16; clinical recovery, March 28; discharged, April 5.

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Case 3: Children's Memorial Hospital, service Dr. George S. Livingstone. Female, age 10 years, admitted, Jan. 18, 1939. Pain in left ear and left neck two weeks with neck tenderness. Fever 99° to 104°, chill second day. Ears negative; spinal fluid normal; Tobey-Ayer test indicates left jugular block. Jugular exposed, appeared normal; blood withdrawn from it sterile. Three days later, X-ray shows coalescent mastoid, left. Mastoidectomy; purulent thrombus in lateral sinus drained; left jugular ligated. Feb. 1, stiff neck, bilateral Babinski. Spinal tap; fluid shows 3,370 cells., polys., 84 per cent; culture shows streptococcus viridans. Sulfanilamide discontinued, sulfapyridine substituted. Temperature reached normal, Feb. 8, but fever recurred. This cycle has been repeated, twice the fever and symptoms abated, the spinal fluid became sterile, but symptoms recurred when spinal drainage was discontinued, and in spite of administration of sulfapyridine. The child is not yet well. The left petrous apex shows slight clouding, and the question arises of advisability of exploratory operation.

Case 4: Admitted to the Illinois Eye and Ear Infirmary with a history of otitis media of 10 days' duration; diagnosis: mastoiditis and meningitis. Mastoidectomy same day; uncovering dura middle and posterior fossa; beginning coalescence, alternating with hard bone. Culture from mastoid, streptococcus hemolyticus; culture from spinal fluid, a Gram positive diplococcus, which I believe to be a mutation form of the streptococcus; further cultures of spinal fluid negative. Sulfanilamide therapy, 8 gm. per day for two days, then gradually reduced. Uneventful recovery.

Case 5: Oak Park Hospital, reported by Dr. John Theobald and Dr. A. H. Parmelee. Male child with otitis media and early meningitis. Culture from spinal fluid reported as diplococci in chains morphologically pneumococcus. Believed to be pneumococcus type III. Mastoidectomy and sulfanilamide therapy. Recovery.

Three patients to County Hospital, who died within 24 hours, diagnosed at postmortem as pneumococcus meningitis.

Four other cases of pneumococcus meningitis, not otogenous origin, sulfapryridine therapy and recovery. I haven't the details on these cases.

According to these cases, sulfanilamide is useful in type III pneumococcus. In other types, sulfapyridine seems to be the remedy of choice, supplemented by the appropriate serum, which, according to some, should also be given intraspinally. The tendency to gastric disturbance is a considerable drawback with this drug. Perhaps rectal or intravenous use of the new sodium sulfapyridine will help. The use of air, a few less cubic centimetres injected than the amount of spinal fluid removed, in Case 2, was suggested by Dr. Brunner, who believes it may have some value in preventing spinal block.

The case of streptococcus viridans infection, on analysis, appears to have had its remissions, due rather to spinal drainage than to the chemotherapy. Incidentally, I know of a case of subacute bacterial endocarditis in which sulfapyridine was tried without success.

A few days before I left Chicago, the streptococcus viridans case (Case 3) was reoperated; the perilabyrinthine cells were found softened and were curetted through a posterior fossa approach; the apex was reached through the carotid canal approach, but no pus found. Nevertheless, there is now considerable discharge from this region, and the child's condition appears better for the time being.

I believe that at present our best guide in the use of chemotherapy is the drug concentration in the blood and the white blood count. The optimum blood concentration apparently differs for different patients and will have to be worked out.

25 East Washington Street.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of April 19, 1939.

SYMPOSIUM: ACUTE CONTAGIOUS DISEASES — EAR, NOSE AND THROAT COMPLICATIONS.

(Continued from July issue.)

(e) Poliomyelitis. Dr. Philip M. Stimson (Continued).

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

Dr. Wilson has given a very detailed and lucid description of acute laryngotracheobronchitis; however, I feel that the subject is so important, and there are so many misconceptions about this disease that it would be worth while to give a few fundamental points about croup in general. When we are confronted with a case of croup, there are two things we think of: 1. Is this a case of croup? 2. What are we going to do for it? In order to establish the diagnosis of croup, we have to find evidence of some mechanical obstruction in the windpipe. All types of cases have been sent into the Willard Parker Hospital diagnosed as croup—poliomyelitis, convulsions, coma, heart failure, pertussia, asthma — everything you can mention. The first thing to establish is the presence of mechanical block in the windpipe. This is evidenced, first, by stridor and, second by retraction of the chest wall with inspiration. I must utter a word of caution here; in infants the chest wall is very, very mobile, and any disorder of the respiration, as occurs in pneumonia, asthma or heart failure, will result in mild retractions. One should not be misled into thinking that the patient has croup just because there are these mild retractions. After the diagnosis of croup is established, we have to decide as to what type of croup it is, and the first thing we do is to find out whether the patient is hoarse. If he can speak, well and good. If not, make the child cry and note whether there is any hoarseness. If the voice is clear, the obstruction is above or below the vocal cords, and one should suspect some other etiology than infection — for example, foreign body, or any one of a number of conditions which may cause stenosis of the windpipe. The parents should be closely questioned as to the possibility of aspiration of a foreign body, whether the child had eczema, or was exposed to any contagious disease, also whether there is asthma in the family. If, on the other hand, the patient is hoarse, the chances are quite likely that you are dealing with a case of infectious croup, and the next question that arises is, Is this a case of diphtheria or nonspecific infection? Unless one is absolutely certain that there has been a negative Schick test in the past six months, every case should be considered diphtheria unless proved otherwise, and I would advise that every case of croup with retractions and hoarseness be admitted to a hospital where proper facilities for treatment of this condition exist. As was mentioned here by Dr. Kramer at one of the recent meetings, stridor should not only be heard but seen, and every case of croup deserves a direct laryngoscopy to determine its cause. In skillful hands, the procedure is harmless and yields a wealth of information necessary in the treatment. We can see whether the obstruction

is due to edema, membrane, mucopurulent exudate, spasm or any combination of these. A direct laryngeal culture should be taken.

A brief review of the living pathology of this condition will be of value. In both diphtheritic and nonspecific croup ,the pathological expression is the same; namely, edema, membrane formation, mucopurulent exudation and spasm. They differ only in frequency and in degree. Thus, edema and mucopurulent secretion are more apt to occur in the nonspecific variety, and membrane in the diphtheritic type; however, I have seen thick, fibrinous membrane in severe streptococcic infections, and only edema or edema with mucopurulent secretion in diphtheria. The final diagnosis rests with the laboratory, and here, too, I would like to caution you to add cultures of the nose and throat for greater accuracy in bacteriological diagnosis.

I won't go into the treatment very much — certainly not the palliative treatment, except to say that one of the most important factors is the attempt to tide over a patient without having to resort to mechanical intervention. There should be, first, very active use of antipyretics, particularly sponges; secondly, administration of oxygen; and lastly, as absolute rest as possible, all treatments to be done at one time. These things cannot be over-emphasized. I have seen many patients admitted to the ward, who, on admission, seemed to be in urgent need of mechanical intervention, but were tided over in this way and made uneventful recoveries; however, these patients must be watched very closely, and the judgment as to how long one can safely wait can be acquired only after long experience.

If mechanical intervention is required there are three methods at our disposal: suction, intubation and tracheotomy. I haven't the time to go into the indications for these, except to say that suction is used when there is something to remove. Dr. Wilson has described the Willard Parker suction tube. With this, rapid and effective aspiration can be performed without the use of a bronchoscope. Intubation is used only when the obstruction is due to subglottic edema. Many men have condemned the use of intubation, and even as recently as last year two papers were published in which the use of intubation is condemned, but suction was not used in conjunction with it. They stated that suction was apparently deserving of a trial. Anybody who uses intubation without suction will have poor results, and these results are without value for purposes of comparing them with results in tracheotomy. By use of suction-intubation, tracheotomy has become an emergency procedure at the Willard Parker Hospital only in cases of supraglottic edema. Dr. Wilson has told you about the other indications in which tracheotomy is performed by choice; namely, in cases where the tube is coughed up or blocked up too frequently, or there is danger of the patient becoming a chronic tube carrier. Supraglottic edema is a very important condition and differs remarkably from subglottic edema. The most striking feature about it is its treacherousness. I have seen patients apparently quite well, breathing with very little difficulty, who in the next few minutes have suddenly suffocated and died. The reason for this is that in supraglottic edema there may be considerable swelling, but the swollen tissues bulge into the pharynx so that there may be very little respiratory obstruction; however, a sudden cough, deep breath or even a change of position may displace these waterlogged tissues so that they are suddenly drawn into the glottis. With each succeeding frantic inspiratory effort, the tissues are jammed down more tightly and the patient may become asphyxiated before a tracheotomy can be done. For this reason, every suspected case of supraglottic edema should be hospitalized at once and a tracheotomy performed as soon as possible, rather than risk sudden asphyxiation. Intubation in these cases is very dangerous because the swollen tissues are displaced around and over the top of the tube so that sudden plugging may ensue. A pathognomonic sign of this condition, which I described some years ago, is the characteristic respiratory sounds. There is a low-pitched inspiratory stertor, followed by a louder and lower-pitched, coarse, expiratory rattle resembling a snore. This sign can be simulated by trying to gargle with the mouth dry. This snore is heard often during sleep, but not when the patient is awake, and if it is

heard in a patient who is awake, the patient should be taken to the hospital at once and a confirmatory laryngoscopy should be performed.

In conclusion, I would simply like to state this: that the treatment of these cases is so specialized, and the prognosis so serious, and the opportunity for acquiring intimate knowledge through personal experience is usually so slim in each individual case that I think it would be wise for general hospitals to assign the care of these patients to a small group of laryngologists and pediatricians, who could thus become more familiar with the special problems encountered in this disease and, therefore, better able to help these children.